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TRANSACTIONS

OF THE

AMERICAN CLIMATOLOGICAL AND CLINICAL ASSOCIATION.

FOR THE YEAR 1915.

VOLUME XXXI.

"The object of this Association shall be the Clinical Study of Disease, especially of the Respiratory and Circulatory Organs, and of Climatology and Hydrology."—*Constitution.*

PHILADELPHIA:

PRINTED FOR THE ASSOCIATION

1915.

NOTICE.

The Association assumes no responsibility for the statements and opinions expressed in the papers read at its meetings.

OFFICERS OF THE ASSOCIATION.

Presidents.

HENRY SEWALL, M.D., Denver, 1915.
JAMES ALEXANDER MILLER, M.D., New York, 1916.

Vice-Presidents.

ARTHUR K. STONE, M.D., Boston, 1915.
PHILIP KING BROWN, M.D., San Francisco, 1916.
HENRY MARTYN BRACKEN, M.D., Minneapolis, 1916.

Secretary and Treasurer.

GUY HINSDALE, M.D., Hot Springs, Va.

Representative to the Executive Committee of the Congress of American Physicians and Surgeons.

THOMAS DARLINGTON, M.D., New York.

Council.

JOHN W. BRANNAN, M.D., New York.
JUDSON DALAND, M.D., Philadelphia.
CHARLES L. MINOR, M.D., Asheville.
JAMES M. ANDERS, M.D., Philadelphia.
HENRY SEWALL, M.D., Denver.

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LIST OF OFFICERS.

Presidents.

NAME	YEAR
A. L. Loomis	1884–5
William Pepper	1886
Frank Donaldson	1887
A. L. Loomis	1888
V. Y. Bowditch	1889
Charles Denison	1890
F. I. Knight	1891
W. E. Ford	1892
R. G. Curtin	1893
A. H. Smith	1894
S. E. Solly	1895
J. B. Walker	1896
E. Fletcher Ingals	1897
E. O. Otis	1898
Beverley Robinson	1899
Abraham Jacobi	1900
Robert H. Babcock	1901
Samuel A. Fisk	1902
Norman Bridge	1903
James C. Wilson	1904
W. F. R. Phillips	1905
E. L. Shurly	1906
Thomas Darlington	1907
Thomas D. Coleman	1908
Charles E. Quimby	1909
Edward R. Baldwin	1910
John Winters Brannan	1911
Alexander D. Blackader	1912
Charles L. Minor	1913
James M. Anders	1914
Henry Sewall	1915
James Alexander Miller	1916

Vice-Presidents.

NAME	YEAR
F. I. Knight, W. H. Geddings	1884-5
Frank Donaldson, Beverley Robinson	1886
V. Y. Bowditch, R. G. Curtin	1887
A. Y. P. Garnett, J. T. Whittaker	1888
J. R. Leaming, E. T. Bruen	1889
A. L. Gihon, H. B. Baker	1890
E. L. Trudeau, T. S. Hopkins	1891
E. Fletcher Ingals, Beverley Robinson	1892
A. H. Smith, E. O. Otis	1893
I. Hull Platt, E. L. Trudeau	1894
John H. Musser, G. R. Butler	1895
Charles E. Quimby, James A. Hart	1896
S. A. Fisk, John C. Munro	1897
Beverley Robinson, C. F. McGahan	1898
James A. Hart, R. C. Newton	1899
R. H. Babcock, J. W. Brannan	1900
Albert C. Peale, S. W. Langmaid	1901
Norman Bridge, W. F. R. Phillips	1902
James C. Wilson, H. S. Orme	1903
Thomas Darlington, Thomas D. Coleman	1904
S. G. Bonney, S. D. Risley	1905
A. D. Blackader, Henry Sewall	1906
Frank Fremont-Smith, C. L. Minor	1907
Judson Daland, Charles Fox Gardiner	1908
E. R. Baldwin, C. E. Edson	1909
James M. Anders, H. Longstreet Taylor	1910
Will Howard Swan, John H. Lowman	1911
Herbert Maxon King, Carroll E. Edson	1912
James M. Anders, C. D. Alton	1913
Lawrason Brown, Will Howard Swan	1914
Arthur K. Stone, James Alex. Miller	1915
Philip King Brown, Henry M. Bracken	1916

Secretaries and Treasurers.

James B. Walker	1884–95
Guy Hinsdale	1895

LIST OF MEMBERS.

Honorary Members.

ELECTED

1888. Abbot, Griffith E., Leominster, Mass.
1914. Fox, R. Fortescue, Devonshire Place, W., London.
1914. Garnett, A. S., Hot Springs, Arkansas.
1914. Gros, Edmund L., 23, Ave. du Bois de Boulogne, Paris, France.
1902. McBride, James H., Pasadena, Cal.
1914. Philip, Sir Robert W., Charlotte Square, Edinburgh, Scotland.
O. M. Reed, Boardman, Alhambra, Cal.
O. M. Robinson, Beverley, 42, West 37th Street, New York.
O. M. Schauffler, Edward W., Kansas City, Missouri.
1911. Stupart, Prof. R. F., Director, Dominion Meteorological Service, Toronto, Canada.
1898. Sunderland, Septimus, 11, Cavendish Place, Cavendish Square, W., London.
O. M. Tyndale, J. Hilgard, Richards Block, Lincoln, Nebraska.
1897. Weber, Sir Hermann, 10, Grosvenor Street, W., London, England.
1907. Williams, Leonard L. B., 123, Harley Street, London, W.

Corresponding Members.

1912. Carpenter, Ford A., Weather Bureau, Los Angeles, Cal.
1898. Eyre, G. G., Claremont, Cape Town, South Africa.
1910. Forbes, Mr. N. Hay, F.R.C.S., Church Stretton, England.
1911. Grenfell, Wilfred, C.M.G., St. Anthony, Newfoundland.

O. M., Original Member.

ELECTED

1912. Henderson, Yandell, 440, Prospect Street, New Haven, Conn.

1908. Liston, Major W. Glen, C.I.E., Indian Medical Service, Director of the Bacteriological Laboratory, Bombay, India.

1910. Rogers, Lieut.-Colonel Sir Leonard, C.I.E., F.R.C.S., Indian Medical Service, Professor of Pathology, Medical College, Calcutta, India.

1904. Sandwith, Fleming Mant, 31, Cavendish Square, W., London.

1902. Weber, F. Parkes, 19, Harley Street, W., London.

1914. Wood, Neville, 42, Elvaston Place, Queen's Gate, London.

Active Members.

1914. Alsever, William D., 727, East Willow Street, Syracuse, New York.

1897. Alton, Charles D., 1310, Asylum Avenue, Hartford, Conn.

1899. Anders, James M., 1605, Walnut Street, Philadelphia.

1890. Anderson, B. P., 1231, N. Cascade Avenue, Colorado Springs, Col.

1900. Arnold, Horace D., 520, Commonwealth Avenue, Boston.

1893. Babcock, R. H. 311, Belden Avenue, Chicago.

1915. Bailey, Walter C., 506, Beacon Street, Boston.

1898. Baldwin, Edward R., Saranac Lake, N.Y.

1901. Barlow, W. Jarvis, Security Building, Los Angeles, Cal.

1907. Barnes, Harry Lee, Wallum Lake, R.I.

1902. Bergtold, William H., 1159, Race Street, Denver, Col.

1897. Blackader, Alexander D., 236, Mountain Street, Montreal, Canada.

1914. Blue, Rupert, Surgeon-General, U.S. Public Health Service, Washington, D.C.

1895. Boardman, W. S., 63, Mt. Vernon Street, Boston.

1897. Bonney, S. G., Stedman Building, Denver, Col.

1885. Bowditch, V. Y., 506, Beacon Street, Boston.

ELECTED

1901. Bracken, Henry Martyn, 1010, Fourth Street, S.E., Minneapolis, Minn.
1891. Brannan, John W., 48, West 51st Street, New York City.
1894. Bridge, Norman, Temple Auditorium, Los Angeles, Cal.
1903. Brown, Lawrason, 104, Main Street, Saranac Lake, N.Y.
1903. Brown, Philip King, 516, Sutter Street, San Francisco, Cal.
1897. Brown, Sanger, Kenilworth, Illinois.
1907. Browning, Charles C., Story Bldg., Los Angeles, Cal.
1909. Bushnell, George E., U.S.A., Fort Bayard, New Mexico.
1886. Butler, G. R., 226, Gates Avenue, Brooklyn.
1914. Byers, John Roddick, Ste. Agathe des Monts, P.Q., Canada.

1896. Campbell, W. A., 424, N. Nevada Ave., Colorado Springs, Col.
1910. Carrington, P. M., U.S.M.H. and P.H. Service, San Diego, Cal.
1898. Casselberry, W. E., 15, East Washington Street, Chicago, Ill.
1906. Claytor, Thomas A., 1826, R. Street, N.W., Washington, D.C.
1901. Cobb, J. O., U.S.P.H. Service, Marine Hospital, Chicago.
1894. Coleman, Thomas D., 505, Greene St., Augusta, Ga.
1901. Collins, Charles Farnham, 50, West 55th Street, New York City.
1889. Coolidge, A., jun., 613, Beacon Street, Boston.
1910. Crane, Bayard T., Rutland, Mass.

1907. Da Costa, John C., jun., 264, South 15th Street, Philadelphia.
1892. Daland, Judson, 317, South 18th Street, Philadelphia.
1890. Darlington, Thomas, 27, Washington Square North, New York City.
1907. Darnall, Wm. Edgar, 1704, Pacific Ave., Atlantic City.

ELECTED

1897. Davis, N. S., 7, West Madison Street, Chicago.
1910. Dixon, Samuel G., Harrisburg, Penn.
1905. Dunn, William LeRoy, Asheville, N.C.

1897. Edson, Carroll E., McPhee Building, Denver, Col.
1915. Elliott, John Barnwell, jun., 608, Maison Blanche, New Orleans, La.
1903. Elliott, J. H., 11, Spadina Road, Toronto, Canada.
1892. Elsner, H. L., Fayette Park, Syracuse, N.Y.

1912. Farrand, Livingston, Boulder, Colorado.
1910. Floyd, Cleaveland, 246, Marlborough Street, Boston, Mass.
1885. Ford, Willis E., 266, Genesee Street, Utica, N.Y.
1911. Fulton, Frank Taylor, 36, Prospect Street, Providence, R.I.

1896. Gardiner, C. Fox, 818, North Cascade Avenue, Colorado Springs, Col.
1892. Gibson, William M., 260, Genesee Street, Utica, N.Y.
1915. Goodman, Edward H., 248, South 21st Street, Philadelphia.
1909. Griffin, Walter Alden, Sharon, Mass.

1907. Hall, J. N., 452, Metropolitan Building, Denver, Col.
1912. Hamman, Louis, 714, Park Avenue, Baltimore.
1893. Hance, I. H., Lakewood, N.J.
1891. Hart, James A., P.O. Box 144, Geneva, N.Y.
1912. Hastings, Thomas W., 172, East 71st Street, New York.
1912. Hatfield, Charles J., 2008, Walnut Street, Philadelphia.
1914. Hawes, John B., 2nd, 29, Gloucester Street, Boston, Mass.
1896. Heffron, John L., 582, South Salina Street, Syracuse, N.Y.
1912. Hewlett, A. Walter, 1835, Cambridge Road, Ann Arbor, Michigan.
1893. Hinsdale, Guy, Hot Springs, Virginia.
1902. Hoagland, Henry W., 818, North Nevada Avenue, Colorado Springs, Col.
1914. Holden, G. Walter, Agnes Memorial Sanatorium, Denver, Colorado.

ELECTED

O. M. INGALS, E. FLETCHER, Monroe Buildings, 104, Michigan Avenue, Chicago.

1889. JACOBI, A., 19, East 47th Street, New York.
1888. JAYNE, W. A., 408-412, Metropolitan Building, Denver, Col.
1901. JENNINGS, CHARLES GODWIN, 457, Jefferson Street, Detroit, Michigan.
1915. JOHNSTON, COLLINS H., 87, Monroe Ave., Grand Rapids, Michigan.

1910. KAHLO, GEORGE D., White Sulphur Springs, West Virginia.
1904. KING, HERBERT MAXON, Liberty, New York.
1907. KINGHORN, HUGH M., 14, Church Street, Saranac Lake, N.Y.
1899. KLEBS, ARNOLD C., c/o J. M. Forbes and Co., Sears Bldg., Boston.
1903. KYLE, D. BRADEN, 1517, Walnut Street, Philadelphia.

1910. LANDIS, H. R. M., 11, South 21st Street, Philadelphia.
1914. LEE, ROGER I., Harvard University, Cambridge, Mass.
1914. LICHTY, JOHN A., 4634, Fifth Avenue, Pittsburg, Pa.
1909. LOCKE, EDWIN ALLEN, 311, Beacon Street, Boston, Mass.
1913. LORD, FREDERICK T., 305, Beacon Street, Boston.
1904. LOWMAN, JOHN H., 1807, Prospect Ave., S.E., Cleveland, O.
1907. LYMAN, DAVID RUSSELL, Wallingford, Conn.

1906. MANGES, MORRIS, 72, East 79th Street, New York.
1902. MARVEL, PHILIP, 1616, Pacific Avenue, Atlantic City, N.J.
1887. MAYS, THOMAS J., 1829, Spruce Street, Philadelphia.
1915. MERCUR, WM. H., 5244, Fifth Ave., Pittsburgh, Pa.
1905. MILLER, JAMES ALEXANDER, 379, Park Ave., New York.
1909. MINER, CHARLES H., 115, South Franklin Street, Wilkesbarre, Pa.
1909. MINER, STANLEY G., 58, Cadillac Square, Detroit, Mich.

ELECTED

1899. Minor, Charles L., Asheville, N.C.
1909. Morgan, James Dudley, 919, Fifteenth Street, N.W., Washington, D.C.

1895. Newton, R. C., 42, Church Street, Montclair, N.J.
1907. Nichols, Estes, 655, Congress Street, Portland, Maine.
1907. Nichols, John B., 1321, Rhode Island Ave., N.W., Washington, D.C.
1912. Norris, George W., 1530, Locust Street, Philadelphia.

1888. Otis, E. O., 381, Beacon Street, Boston.

1913. Parfitt, C. D., Gravenhurst, Ontario, Canada.
1912. Paterson, Robert Childs, Saranac Lake, New York.
1906. Perkins, Jay, 125, Waterman Street, Providence, R.I.
1895. Phillips, W. F. R., School of Medicine, Charleston, S.C.
1887. Platt, Walter B., 802, Cathedral Street, Baltimore.
1902. Pottenger, F. M., 1100, Title Insurance Building, Los Angeles, Cal.
1905. Pratt, Joseph H., 317, Marlborough Street, Boston.
1914. Probst, Charles O., 185, State Street, Columbus, Ohio.
1905. Pryor, John H., 26, Linwood Ave., Buffalo, N.Y.

1891. Quimby, Charles E., 278, West 86th St., New York.

1885. Rice, C. C., 123, East 19th Street, New York.
1901. Richardson, Charles W., 1317, Connecticut Avenue, Washington, D.C.
1904. Richer, A. J., Ste. Agathe des Monts, Province of Quebec, Canada.
1890. Robinson, W. D., 2012, Mt. Vernon St., Philadelphia.
1902. Rochester, Delancey, 469, Franklin Street, Buffalo, N.Y.
1892. Roe, John O., 44, South Clinton St., Rochester, N.Y.
1890. Rogers, E. J. A., 222, Colfax Avenue, Denver, Col.

1905. Schauffler, William Gray, 400, Madison Ave., Lakewood, N.J.
1901. Sewall, Henry, 1360, Vine Street, Denver, Col.

ELECTED

1915. Shattuck, George Cheever, 205, Beacon Street, Boston.
1910. Shurly, B. R., 32, Adams Avenue W., Detroit, Mich.
1890.*Smith, A. Alexander, 18, West 51st St., New York.
1887. Smith, Frank Fremont, 1808, Massachusetts Ave., Washington, D.C.
1911. Steiner, Walter R., 4, Trinity Street, Hartford, Conn.
1909. Stevens, Martin L., Asheville, N.C.
1915. Stone, Willard J., Colton Building, Toledo, Ohio.
1904. Stone, Arthur K., 44, Fairfield St., Boston, Mass.
1910. Swan, John M., 457, Park Avenue, Rochester, N.Y.
1901. Swan, Will Howard, 1440, North Nevada Avenue, Colorado Springs, Col.

1892. Taylor, H. Longstreet, 75, Lowry Arcade, St. Paul, Minn.
1907. Taylor, J. Gurney, 514, Goldsmith Building, Milwaukee, Wisconsin.
1896. Taylor, J. Madison, 1504, Pine Street, Philadelphia.
1910. Thomas, John D., 1726, M. Street, N.W., Washington.

1897. Whitcomb, H. H., 622, Dekalb St., Norristown, Pa.
1911. White, William Charles, Bedford Ave. and Wandless St., Pittsburg, Pa.
1898. Whitney, Herbert B., 320, Temple Court, Denver, Col.
1898. Williams, Harold, 528, Beacon Street, Boston.
1885. Williams, H. F., 416, Grand Avenue, Brooklyn.
1911. Williams, Linsly R., 882, Park Avenue, New York City.
1910. Wilson, Gordon, 4, East Preston St., Baltimore.
o. m. Wilson, James C., 1509, Walnut Street, Philadelphia.
1913. Wood, Nathaniel K., 309, Beacon Street, Boston.

Total, 138 Active Members.

* Died November 13, 1915.

MINUTES OF THE THIRTY-SECOND ANNUAL MEETING OF THE AMERICAN CLIMATOLOGICAL AND CLINICAL ASSOCIATION.

June 18-19, 1915.

The meeting convened June 18, 1915, at 10 o'clock, at t' Hotel Clift, San Francisco, California, the President, L.. Henry Sewall, in the chair. The Secretary being absent, Dr. Pottenger acted as Secretary *pro tem*. The following scientific programme followed :—

(1) President's Address. Dr. Henry Sewall.

(2) Memoirs of Dr. Samuel A. Fisk and Dr. Samuel W. Langmaid.

(3) "The Importance of the Personal Equation in the Treatment of Tuberculosis." By Dr. W. H. Swan. This paper was read by Dr. Holden, Dr. Swan being absent. It was discussed by Drs. Sewall, Hall, Brown, Barlow, J. M. Swan, Colonel Bushnell, and Dr. Holden.

(4) (*a*) "A Case of Coccidioidal Granuloma." (*b*) "Cases of Unusual Aneurisms." By Dr. Philip King Brown. Discussed by Dr. Dixon, Associate in Pathology, Stanford University, California, and Dr. Cummins, of the Southern Pacific Hospital (on invitation).

(5) "The Blood-pressure in Cases of Dysthyroidism." By Dr. J. M. Swan. Discussed by Drs. Sewall, Lichty, Barlow, Brown, and Swan.

Business Meeting.

Calling of the roll showed thirteen active members, one corresponding member, and one honorary member present.

The minutes of the previous meeting were read and adopted.

The report of the Secretary and Treasurer was received and placed on file. An Auditing Committee consisting of

Drs. Holden and J. M. Swan were appointed to audit the Treasurer's report.

Colonel Bushnell, Drs. Hall, Campbell, Barlow, and Browning were appointed as the Nominating Committee.

The report of the Council recommending the following names for membership was read: Drs. Geo. C. Shattuck, Walter C. Bailey, John Barnwell Elliott, Willard J. Stone, Collins H. Johnston, Edward H. Goodman, Wm. H. Mercur.

June 18, 2 *p.m.*

The Society resumed its scientific programme.

(1) "The Physician and the Weather Bureau." By Ford Carpenter. Discussed by Drs. Sewall, Barlow, Lichty, Browning, and Mr. Carpenter.

(2) "Lung Involvement Secondary to Suppurative Abdominal Diseases." Dr. J. N. Hall. Discussed by Drs. Sewall, Lichty, Colonel Bushnell, Drs. Brown and Hall.

(3) "The Acute Infections in Diabetes Mellitus." By Dr. John A. Lichty. Discussed by Drs. Sewall, Pottenger, and Lichty.

(4) "Case of Pulmonary and Glandular Aspergillosis." By Dr. Walter G. Holden. Discussed by Drs. Sewall and Browning.

June 19, 10 *a.m.*

(1) "Cardiac Syphilis: Its Incidence and Diagnosis." By Dr. James M. Anders. In the absence of Dr. Anders the paper was read by Dr. J. N. Hall. Discussed by Drs. Sewall, J. M. Swan, Lichty, Sewall, McBride, Hall, and Pottenger.

(2) "Some of the Disputed Anatomic-pathologic Questions in Tuberculosis." By Dr. F. M. Pottenger. Discussed by Drs. Sewall, Otis, McBride, Colonel Bushnell, Drs. Swan and Pottenger.

Business Meeting.

June 19, 12 *noon.*

Dr. Holden reported for the Auditing Committee that the books and vouchers had been examined and found correct.

Colonel Bushnell reported for the Nominating Committee recommending the following officers for the coming year:—

President: Dr. James Alexander Miller, New York.

Vice-President: Dr. Philip King Brown, San Francisco.

Second Vice-President: Dr. H. M. Bracken, Minneapolis.

Secretary and Treasurer: Dr. Guy Hinsdale, Hot Springs, Virginia.

Member of Council: Dr. Henry Sewall, Denver.

Committee of Arrangements for the Congress of Physicians, to be held in Washington, 1916: Dr. Chas. W. Richardson, Dr. F. Fremont Smith (alternate).

The Secretary was instructed to cast the ballot for the nominees as reported by the Nominating Committee. The names which had been recommended for membership by the Council were elected unanimously.

The following resolutions calling for changes in the constitution were unanimously adopted:—

Resolved: That Section 1, Article IV, of the Constitution be changed by inserting after the word "Treasurer" the words "and a Recorder."

Resolved: That Section 4, Article IV, be changed by inserting after the word "Treasurer" the words "and Recorder."

Resolved: That Article V of the Constitution be changed by striking out the words "He shall superintend the publication of the Transactions under the direction of the Council," and insert at the end, so as to constitute Section 3, the words "Section 3. *Recorder.* The Recorder shall secure the papers read and also proper notes of the discussions thereon and shall superintend, under the direction of the Council, the publication of the Transactions."

The Nominating Committee proposed the name of Guy Hinsdale for Recorder. The Secretary was instructed to cast the ballot for Dr. Hinsdale.

Adjournment.

A VISIT TO AREQUIPA SANATORIUM.

Those of us who attended the thirty-second meeting of the Association in San Francisco felt well repaid for the output of time, labour, and money. California, apparently, has a population whose greatness of heart is no less unique than her storied climate.

After a scientific session of brief but vivacious interest, on June 20, the day following its close, the Association, together with a number of interesting laymen, became the guests of our Philip King Brown in a luncheon at the Arequipa Sanatorium, after which we enjoyed that marvellous picture show seen in a ride to the top of Mount Tamalpais. Ferry across the bay of San Francisco, a ride by rail through a country of flowers and a suggestion of subtropical vegetation, with the finishing half-mile by automobile brought us in less than two hours to the Sanatorium.

The place has a rugged sylvan charm. The buildings perch upon a steep hillside and look out towards the east over a vast amphitheatre of green wood and brush. Their rough board structure harmonizes with the environment and strengthens the illusion that here is a camp where one comes to drop care and pick up health. The sleeping quarters are great halls opening broadly on verandahs.

The policy of the institution makes it somewhat unique among its kind. Admission to its care is limited to the working girls of San Francisco, forty thousand strong, among whom the death-rate from consumption is twice as high as among the men.

It is sought to cull from the applicants those in the early stages of the disease and to maintain them at the cost of a dollar a day. An important part of the design is to offer to the patients some work which may be carried on without injury to health and prove of sufficient commercial value to bring them financial returns. For patients confined to bed the charge is one dollar and a half per day.

The third annual report of the Sanatorium was issued in September, 1914, and it appears that the plans on which the project was organized have been adhered to with great fidelity. Up to the date mentioned there had been treated at Arequipa 179 patients, twenty-five of whom still remain in the Sanatorium.

The making of high-grade art pottery has been chosen as the elective occupation of the institution. The product of the pottery is practically the work of the patients, except the digging and the screening of the clay, the glazing and preparing the kiln for firing. No patient is allowed in the pottery who coughs or has a temperature above normal. To those patients who choose the work some simple task is given at the outset, lasting not more than an hour a day, and none work more than five hours a day, five days in the week. Although the pottery establishment has not, as a whole, thus far paid for itself, and is still in an experimental stage, the outlook is encouraging. Some of the patients have been paid as much as fifteen dollars a week for their work. No harm has been traced to the occupation.

We visitors were impressed with the happy, blooming faces of the girls under treatment, and we could not but reflect on the greatness of accomplishment and how it might be magnified did more of us possess the initiative and devotion of Philip King Brown.

H. S.

SAMUEL A. FISK, M.D.

President, 1902.

IN MEMORIAM.

SAMUEL AUGUSTUS FISK, A.M., M.D.

Since its last meeting our Association has lost one of its most loyal and lovable members.

Samuel Augustus Fisk died in Boston, January 18, 1915.

He was born in Northampton, Massachusetts, February 9, 1856, the son of Robert and Narcissa Perry (Whittemore) Fisk. His father died when he was a child, and he was brought up by his uncle, Samuel Augustus Fisk, M.D. (Yale, 1844), a well-known and honoured physician of Northampton, after whom he had been named.

He graduated A.B. from Yale in the class of 1877, and M.D. from Harvard in 1880. He had just entered his service as house officer in the Massachusetts General Hospital when an attack of tuberculous pneumonia obliged him to go west for his health. After a trial of various places, of which he gave this Society an interesting account in the *Transactions* of the year 1907, he settled in Denver, Colorado, in 1883, where he soon built up a large and successful practice. He was always interested in the advancement of medical interests in Denver and Colorado, and was President of the Colorado State Medical Society in 1888. He took an active part in bringing the American Medical Society to Denver for its meeting in 1898; and besides hard work on the Finance Committee of Arrangements, was Chairman of the Section in Medicine of the Association. It is really from this meeting that the new era of the American Medical Association dates.

In Denver, Dr. Fisk took, from the first, an active and useful part in medical education. He was made Professor of Medicine in the Medical School of the University of Denver in 1884, and was Secretary of the School until 1895, when he was chosen Dean, in which office he served until

1899. He was one of the founders and most liberal supporters of the Medical Library, now the property of the Medical Society of the City and County of Denver, giving freely of his means and labour towards its establishment. He was a member of the staff of St. Luke's and the County Hospital, and a member of the first board of the Oakes Home. He received the degree of A.M. from Yale in 1884. He was a member of the Association of American Physicians from 1890; a member from its foundation of the National Association for the Study and Prevention of Tuberculosis. In 1899 a nervous disorder, which had foreshown its coming for some time, necessitated his relinquishing active practice in Denver, and after some years of travel he settled in 1905 in Brimfield, Mass., where he made his home. He kept, during a long and trying invalidism, an unbroken interest in medical affairs, and in all matters of public welfare.

He was married in 1905 to Miss Clara Crumb, of Chaumont, N.Y.

His interest was, from the first of his residence in Colorado, turned strongly to climatology, and in that field and in the study and care of tuberculosis his chief work was done. His reputation so made was wide and honoured. His work, writings, and advocacy played a large and influential part in establishing the repute and recognition of Colorado as a health resort.

Dr. Fisk became a member of the Association in 1887, and was its President in 1902. He was a faithful attendant on its meetings, and a frequent contributor to its *Transactions;* but he gave more than his time or thought to our meetings, he gave his loyalty and his love—priceless additions to that store of friendship which has been the characteristic of the Climatological Association.

Keenly interested and skilled in his medical art, Dr. Fisk had a large practice, and of few physicians could it be said so truly that every patient became his friend, for to all he was from the first a friend—true and faithful to the end.

Dr. Fisk had a wide outlook on life, for he was possessed of a keen intellect and his culture had been broad and finished. If in the modern sense he was not a highly scientific physician, he had that which, with his medical skill, was of far greater worth and rarity—a heart overflowing with love for his friends and fellow-men. His altruism was simple, un-

affected, and joyous. Genial and sweet-tempered under conditions of personal discomfort most trying to bear, he was eager to lift the burden from those in ill-health and to inspire hope in those who had to start life anew.

This generous care for others tinged all his acts and thoughts.

This Association owes to him much of its habit of unjealous friendships. In his President's address in 1902 he set forth the ideals for his beloved Climatological Association —work for others, comradeship, and loyalty to truth.

"We are not students of climate from an indifferent, impersonal standpoint. It is our aim to apply to the individual the results of our study. . . . Man has ever been our object of consideration.

"It is our delight to talk of fresh air, to walk by the shore and hear the waves beat, to let the mountain breezes blow through our locks, to remember that God made us human beings to walk in sunshine, to live, and not to starve.

"This gift of life is not limited to any one region, is not confined to any one spot. Man can live north, east, south, and west. One place has characteristics that another has not, and to which man must adapt himself; but wherever he lives, and wherever he makes his home, Truth, the touchstone of science, is one and the same. She was there before man came, she will be there when man leaves.

"Every day the sun, and after sunset, night and her stars.

"It is because we have these views in common that we have come together into an Association."

May this Society keep ever faithfully these ideals of a generous and loyal member.

C. E. E.

SAMUEL WOOD LANGMAID, A.M., M.D.

Dr. Samuel Wood Langmaid died in Brookline, February 3, 1915, in his 78th year. He had been in failing health for the last few years. He was born in Boston, in Charles Street, June 26, 1837. His parents, Samuel H. and Dorcas Sawyer Langmaid, later moved to Roxbury, and in 1855 to Cambridge.

Dr. Langmaid fitted for college at the Roxbury Latin School, and was a member of the Harvard Class of 1859. A year after graduation from college he entered the Harvard Medical School, completing his course as surgical house officer at the Massachusetts General Hospital, and graduating in 1864, with the degrees of A.M. and M.D. In June, 1864, he volunteered as Acting-Assistant Surgeon in the United States Army, and was ordered to White House Landing, Va., but the next month he was sent to the hospital at Readville, Mass., where he served until the closing of the hospital a year later. After a few months in Europe he began general practice in Boston in Park Square. He was on the surgical staff of the Carney Hospital from 1868 to 1880. In 1866 the Children's Hospital was founded and he became one of the surgeons, serving until after the new building in Huntington Avenue was opened in 1882. He then gave up general surgery and established a department for diseases of the throat, of which he had charge for many years.

When Dr. Langmaid began practice the speciality of laryngology was slowly developing in Europe. On account of his own voice and his association with singers, he followed with interest everything connected with the larynx and became one of the pioneers of the speciality in this country. He joined the American Laryngological Association in 1880, two years after its foundation, and was President in 1891, when the Association met in Boston. In 1881 he was appointed assistant physician for diseases of the throat in Dr. Knight's Clinic at the Massachusetts General Hospital, and took charge of this clinic for six months in every year until 1892, when he resigned. He became a member of the American Climatological Association in 1887, and was Vice-President in 1901. He was a member of the Massachusetts Medical Society, and of other medical societies and clubs in Boston. His medical career covered the period of half a century. His private practice became more and more limited to the upper respiratory tract, and after about 1880 he gradually withdrew from other work. He had many patients among the musical and theatrical professions and was often consulted by visiting singers and actors. During the latter years of his life his health became poor, and he finally retired from practice in 1912. He occasionally con-

tributed to the literature of the larynx. He had three papers in the old *Archives of Laryngology,* which in 1880 started its short-lived career. These papers were on "The Treatment of Certain Forms of Vocal Disability by the Application of the Principles of Vocal Culture," "On the Singing Voice," and "A Common Form of Vocal Disability." He later published a dozen or more other articles, which are listed in the *Transactions* of the American Laryngological Association for 1898 and 1908.

Dr. Langmaid did much for vocal music in Boston. He had a beautiful tenor voice and he took every opportunity to promote good singing. While in college he sang in the college chapel, and was leader of the Glee Club. So irrepressible was his music that he was once summoned before the authorities for singing as he walked through the yard. In the course of his life he became a member of many musical associations, the Chickering Club, the Parker Club, the Boylston Club, and later the Apollo Club and the Cecilia. Of his many interests, perhaps the one nearer to his heart than any other outside of medicine was the Harvard Musical Association, of which he was President for many years. He was also a director of the New England Conservatory of Music. For a quarter of a century he sang tenor in the quartet at Trinity Church, before the establishment of the vested choir. He had a large acquaintance not only among the musical people of Boston, but also among the noted singers of the world, several of whom were at times his patients.

His interests and his friendships were not confined to medical and musical circles. As a boy he spent his summer vacations in New Hampshire, and acquired a true sportsman's love for the woods, and hunting and fishing. In later life, whenever he had the opportunity, he went off with his dogs into the country, or with his rod to some trout stream. He thoroughly enjoyed the life in the open, and the intelligence of his dog and the fine art of fishing. He was always a sociable and congenial companion. He was a charter member of the Tavern Club, and for some time a member of the St. Botolph Club. Dr. Langmaid married in 1870 Miss Ella M. Tuttle, and had two daughters, Miss Bertha Langmaid and Mrs. Conrad Hobbs, all of whom survive him.

JOHN HENRY HUDDLESTON, M.D.

Dr. John Henry Huddleston, a member of the American Climatological and Clinical Association since 1905, died in New York City, October 30, 1915, of pneumonia. Dr. Huddleston was a most valued and respected member of this Association and his untimely death, coming at the very prime of his life, is a very severe loss to the Society and a cause of great sorrow to his numerous friends in it.

It is, however, in New York City, where Dr. Huddleston's main activities lay, that his loss is most felt. He had become one of the most public-spirited members of the profession, able in counsel, unsparing in energy, and devoted in service to the public health interests of New York.

He was one of the most instrumental leaders in founding the Association of Tuberculosis Clinics, and as its Vice-President, as a Trustee of the New York State Hospital for Incipient Tuberculosis, and as a Director of the Metropolitan Life Insurance Company, particularly in charge of its anti-tuberculosis work, he has done as much as any one man to further the prevention of tuberculosis in New York.

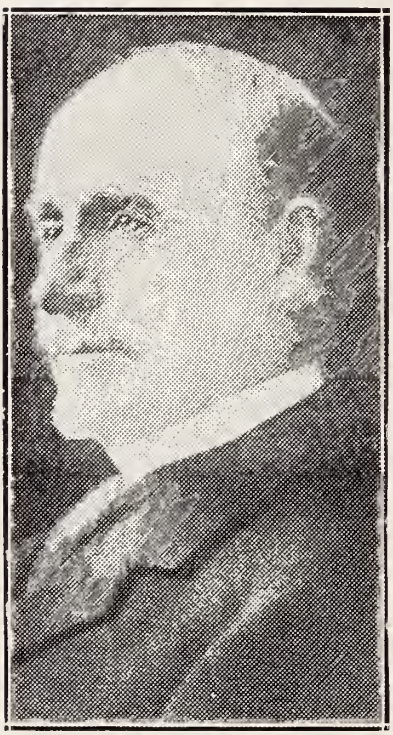
More recently, as a very active member of the Public Health Committee of the New York Academy of Medicine, of the Advisory Council of the New York City Department of Health, and as President of the Public Health Council, a volunteer conference representative of all organizations directly interested in public health, he has exerted a very wide influence upon the active application of the principles of preventive medicine in New York.

Dr. Huddleston was also Visiting Physician to the Gouverneur Hospital, Willard Parker Hospital and Riverside Sanatorium. He was also Consulting Physician to the United Hospital at Portchester, New York.

In his hospital work and private practice he was generally recognized as a man of wise judgment and great skill.

To his personal friends, however, he will chiefly be remembered for his calm, modest, and lovable personality, combined with a faithfulness to duty and to principle which is rarely excelled. J. A. M.

[Dr. John O. Roe died December 24, 1915.]



DR. JOHN O. ROE.

Born February 3, 1849. Died December 24, 1915.

DR. JOHN O. ROE.

Dr. John O. Roe, who was elected an active member of this Association in 1892, died at his home in Rochester, New York, on December 24, 1915, of pernicious anæmia, in his sixty-seventh year. He was of English ancestry, and was descended from John Roe, who settled in Providence, Rhode Island, in 1640. An ancestor, Austin Roe, was a major in the continental army, and his sword was among Dr. Roe's possessions. Dr. Roe was born at Patchogue, Long Island, was educated at the Hudson River Institute and at Wilbraham Academy, Massachusetts, and he received his degree in medicine at the University of Michigan in 1870. He was also graduated in medicine from the College of Physicians and Surgeons in New York City in 1871, receiving the prize for his thesis. After practising for a short time in Rochester, he continued his studies in Berlin and London. Here he was a pupil of Sir Morell Mackenzie, who later, in recognition of Dr. Roe's achievements, proposed his name for membership in the British Medical Association, an unusual honour for one not a citizen of Great Britain or one of her Colonies.

Dr. Roe was the author of numerous papers on laryngology. Among them are "Recurrent Papilloma of the Pharynx" in "Illustrated Medicine and Surgery," 1884; "Injuries and Deformities of the Throat"; "Operations in the Upper Air Passages" in "The American Text-book of Diseases of the Eye, Ear, and Throat," 1899; and various papers contributed to the Transactions of the American Laryngological Association, of which he was the President in 1898. Dr. Roe was for many years laryngologist of the Rochester General Hospital. He was President of the Medical Society of the State of New York, of the Central New York Medical Association, of the Monroe County Medical Association, and of the Rochester Academy of Medicine, of which he was one of the founders.

Dr. Roe was also a member of the Rochester Pathological Society, the Seventh International Medical Congress in London, England; the Eighth International Medical Congress in Copenhagen, Denmark; the Ninth International

Medical Congress in Washington, D.C.; the Tenth International Medical Congress in Berlin, Germany; the Pan-American Medical Congress in Washington, D.C.; a corresponding member of Société Française d'Otologie, de Laryngologie et de Rhinologie, a member of the British Medical Association, American Laryngological, Rhinological and Otological Society, the New York Academy of Medicine, and the Congress of American Physicians and Surgeons.

He was also a fellow of the American College of Surgeons.

Of all honours received by Dr. Roe none touched him more deeply or gave him greater satisfaction than the degree of LL.D., bestowed upon him by his Alma Mater, the University of Michigan, in 1913.

It was during the last fifteen years of his life that Dr. Roe perfected his remarkable technique in the correction of nasal deformities by subcutaneous operations. His paper on this subject, read at the meeting of the Laryngological, Rhinological and Otological Society at Pittsburg in 1908, and his earlier contribution to the same subject, read at the meeting of the Medical Society of the State of New York in 1905, were contributions of marked originality, and with his other papers on "Palato-pharyngeal Adhesions" and on "Methods of Opening the Maxillary Antrum" gave him a wide reputation in this difficult field. His office was thronged with patients from this country and from distant lands seeking relief.

Dr. Roe was singularly modest about his own attainments; he was a genial friend, and left a devoted circle in his own city and in the many societies to which he belonged.

His widow and a brother survive him.

CONSTITUTION AND BYE-LAWS.*

CONSTITUTION.

Article I.—Name.

This Society shall be known as the American Climatological and Clinical Association.

Article II.—Object.

The object of this Asociation shall be the Clinical study of *Disease, especially of the Respiratory and Circulatory Organs, and of Climatology and Hydrology.*

Article III.—Membership.

Section 1.—This Association shall consist of *active*, *corresponding*, and *honorary* members, the former not to exceed 150 and the latter not to exceed twenty (20).

Section 2.—Names of candidates for active membership, whose applications shall have been endorsed by *three* (3) active members, shall be sent to the Secretary at or before the annual meeting at the second business session of which they shall be read and then lie over until the next annual meeting, when such as are approved by the Council shall be balloted on. Three (3) black balls shall be sufficient to reject a candidate. The Council shall have power to nominate active members.

Candidates for membership shall, at the discretion of the Council, present a paper to the Association showing clinical study of merit.

Section 3.—The power of nominating honorary and corresponding members shall be vested in the Council. The election shall be conducted in the same manner as that for active members. Honorary members shall enjoy all the privileges, but shall not be allowed to hold any office or cast any vote.

* As amended June 19, 1915.

Section 4.—Any member of the Association absent from the meetings, in person or by contributed paper, for three (3) consecutive years, without sufficient cause, shall be dropped from the list of members by vote of the Council.

Article IV.—Officers.

Section 1.—The officers of this Association shall consist of a *President*, two *Vice-Presidents*, a *Secretary and Treasurer*, and a *Recorder*, who, with five other members, shall constitute the *Council* of the Association.

Section 2.—*Nominations.* The officers, including the Council, shall be nominated by a committee of five (5) members, which committee shall be nominated by the President at the first session of each annual meeting and shall report at the business meeting.

Section 3.—*Elections.* The election of officers shall take place at the business meeting. A majority of votes cast shall constitute an election.

Section 4.—The President, Vice-Presidents, Secretary and Treasurer, and Recorder shall enter upon their duties at the close of the annual meeting at which they are elected, and shall hold office until the close of the next annual meeting, or until their successors are elected.

Section 5.—Members of the Council, other than the President, Vice-Presidents, Secretary and Treasurer, shall hold office for five (5) years.

Section 6.—*Vacancies.* Any vacancy occurring among the officers of the Association during the year may be filled by the Council.

Article V.—Duties of Officers.

President and Vice-Presidents.

Section 1.—The President and Vice-Presidents shall discharge the duties usually devolving upon such officers. The President shall be *ex-officio* Chairman of the Council.

Secretary and Treasurer.

Section 2.—As Secretary, he shall attend and keep a record of all the meetings of the Association and of the Council, of which latter he shall be *ex-officio* Clerk. At each annual meeting he shall announce the names of all who have ceased to be members since the last report. He shall notify candidates of

their election to membership. He shall send a preliminary notification of the annual meeting two (2) months previous thereto, and the programme for the annual meeting at least two (2) weeks previous to its assembly, to all the members of the Association. He shall also send notification of the meetings of the Council to the members thereof. At each annual meeting of the Association he shall read the minutes of the previous meeting and of all the meetings of the Council that have been held during the current year.

Section 3.—As Treasurer, he shall receive all moneys due, and pay all debts therewith. He shall render an account thereof at the annual meeting, at which time an auditing committee shall be appointed to report.

Recorder.

Section 4.—The Recorder shall secure the papers read and also proper notes of the discussions thereon and shall superintend, under the direction of the Council, the publication of the TRANSACTIONS.

ARTICLE VI.—COUNCIL.

The Council shall meet as often as the interests of the Association may require.

Four (4) members shall constitute a quorum.

It shall have the management of the affairs of the Association, subject to the action of the Association at its annual meetings.

It shall consider the claims of candidates recommended to it for admission to membership.

It shall not have the power to make the Association liable for any debts exceeding in total one hundred dollars ($100), in the course of any one year, unless specially authorized by a vote of the Association.

It shall have the entire control of the publications of the Association, with the power to reject such papers or discussions as it may deem best.

It shall have power to nominate active members at the annual meeting.

The Council shall have power to invite any gentleman, not a member, to read a paper at the annual meeting, on any subject within the scope of the objects of this Association.

The Council shall determine questions by vote, or—if demanded—by ballot, the President having a casting vote.

The Council shall constitute a Board of Trial for all offences

against the Constitution and Bye-laws, or for unbecoming conduct, and shall have the sole power of moving the expulsion of any member.

The President, or any two members, may call a meeting, notice of which will be transmitted to every member two (2) weeks previous to the meeting.

Article VII.—Papers.

Section 1.—The titles of all papers to be read at any annual meeting shall be forwarded to the Secretary not later than one (1) month before the first day of the meeting, in order to appear on the printed programme.

Section 2.—No paper shall be read before the Association which has already been printed or been read before another body.

Article VIII.—Quorum.

A quorum for business purposes shall be ten (10) members.

Article IX.—Amendments.

This Constitution may be amended by a four-fifths ($\frac{4}{5}$) vote of all the members present at an annual meeting, provided that notice of the proposed amendment has been printed in the notification of the meeting at which the vote is to be taken.

BYE-LAWS.

(1) Meetings of the Association shall be held annually.

(2) The time and place of the meetings shall be determined by the Council.

(3) The dues of active members shall consist of an annual assessment not to exceed seven and a half ($7.50) dollars. Members in arrears shall not be entitled to vote. Those in arrears for two (2) years may be dropped from membership by recommendation of the Council.

(4) Order of business meeting.

First day:—

Calling the roll of members.
Minutes of previous meeting.
Secretary's and Treasurer's reports.
Recorder's report.
Report of Committee of Arrangements.

Appointment of auditing committee.
Appointment of nominating committee.
Report of Council on recommendations for membership.

Second day, Morning session:—

Reports of nominating committee and auditing committee.
Election of officers.
Election of members.
Nomination of candidates for membership.
Report of committee on health resorts.
Miscellaneous business.
Adjournment of business meeting.

PRESIDENT'S ADDRESS.

By HENRY SEWALL, Ph.D., Sc.D., M.D.
DENVER, COLORADO.

The thoughts of one appointed to the high position with which you have honoured me inevitably centre upon the means of promoting the welfare of the Association which he represents. As Nature devotes infinite pains to the preservation of her types while reckless of the interests of the individual, so he who has at heart the highest destiny of a society like ours must be guided by laws of intellectual supply and demand unhampered by personal prejudice or temporary expediency.

A living organism represents a moving equilibrium which is only preserved by its ceaseless adjustment to multitudinous varying forces which tend to bring it to rest, which is death; likewise, the price of animation in any organization is the continuous co-ordinated striving of its individuals.

The noble scheme conceived by the founders of this Society, to bring together a band of workers who should systematically investigate the physical forces of Nature as they influence the physiological reactions of the body, does not seem to have been sufficient to maintain the enthusiasm of our body.

A year ago it was formally decided that the best interests of our organization demanded an extension of its scope to embrace practically all conceivable disorders of the human frame. Like pampered children we reject half-eaten our

piece of bread and butter to grab the cake and candy left within our reach. If we are not careful there will be a case of dyspepsia which will need a course of starvation and a return to a kitchen garden regime to cure. Has the scope of legitimate activity laid down by our founders so narrowed that we have no longer sufficient standing room within it, or, rather, has the intellectual curiosity and energy of this generation retrograded, and do we fail to grasp the opportunities spread before us? When this Society was founded radium had not been discovered, and radio-activity had no more practical importance than the pith ball experiment of the lecture room. Now there is a trumpet call for investigators in the physiological and therapeutic bearings of these marvellous energies.

Whose business is it, if not ours, to develop the truth in the claims of those who find mysterious powers as vital stimuli in radio-active waters, high-frequency currents, and the what not fairy tales that come to us from the physical laboratory and the surgical clinic? Has our knowledge of the physiology of climate essentially advanced in the past quarter of a century? It has been demonstrated, indeed, that the supposed inability of the white man to live in the Tropics is more an incident of parasitism than of climatology. It has been shown that the cure of tuberculosis is a question of physiological reactivity to the proper use of climate; but have we not been too well content with the thought that the patient need only move from one side of a wall to the other, from the inside of a house to the outside, to get this needed change, while still enjoying the advantage of our personal ministrations?

How much advanced beyond that of Aristotle is our knowledge of why a muscular rheumatism may fade away on a journey? Is it not time that we had a text-book which

may deal with something like mathematical accuracy on the relations of climate to health and disease? One of the few laws of vital action accepted by physiologists is the generalization of du Bois Reymond to the effect that the stimulating property of any agent is a function of the rate of change in intensity of the stimulus. In climatic change we vary every stimulus acting upon the mind and body of man, and herein may lie the basis of the vital influence of unaccustomed scenes.

The knowledge that the open air is an indispensable entity in the preservation of health and the cure of certain diseases has properly magnified the importance of ventilation; yet, in spite of all research, we remain in the dark as to the biological differences between good and bad air; whether the vital effects of the medium depend wholly on temperature and humidity, or also involve the chemical effects of expired poisons.

As climatology treats of an external environment reaction to which means so much for the welfare of our minds and bodies, so there is an internal environment whose study is still more important, and which, it appears to me, comes definitely within the spirit of the scope originally marked out for the activities of this Society.

I speak of food and its metabolism. An Arctic explorer said that, though he might feel insulted if offered a tallow candle as an *entrée* on the menu of a banquet extended to him at home, the same morsel set before him within the Arctic Circle would be acknowledged with smiling thanks. If it were necessary to banish from the medical curriculum every subject but one, I suspect that our practical usefulness as physicians would be best conserved if that one subject were Dietetics. What more wonderful remedial effects do we witness than those following the proper management of

diet? The meaningless and tiresome aspect which the subject had always presented has been utterly transformed in the light of recent conceptions of intermediary metabolism, and especially of the still mysterious activities involved in the reactions of anaphylaxis. We have learned that the extraordinary manifestations of idiosyncrasy to foods and drugs are only extreme illustrations of forces common to all of us.

We gather from the physiological chemist that the humoral internal environment in which our living cells exist must be maintained at a certain balance of acid and alkali, expressed by the hydrogen-ion content of the blood. Disorders of metabolism which transcend the analysis of the most expert pharmacologist can yet often be foreshadowed or diagnosed by the every-day practitioner who but makes the simple test for the presence of acetone in the urine.

Acidosis, whatever its origin, is an expression of pathologic condition which it behoves the clinician to recognize, for it shows the presence of a criminal agent in the peaceful community of cells, and not infrequently it suggests the photograph and thumb prints of the felon. The practitioner who will make routine tests for acetone in the urine of his patients will see many obscure conditions illuminated, to the improvement of his therapeutic aim.

From reports in the medical press, combined with personal experience, it has seemed to me that there is, as it were, an epidemicity in the condition of acidosis which deserves definite investigation in order to determine the factors, seasonal, climatic, dietetic, or biologic, involved in its occurrence.

I venture to assert that the scientific study of diet has hardly begun. Only a few weeks ago Allen and Joslin independently reported their astonishing conclusions that in

cases of diabetes mellitus, even in the young, the symptoms would ameliorate, the urine become free from sugar, and practically so from acetone, by a course of complete abstinence from food, lasting in cases for a considerable number of days. With careful administration of proper nourishment this desirable condition was afterwards maintained, and the sick one restored to activity and happiness.

My animadversions are not made from the standpoint of the pharmacologist or of the expert in nutrition, but from that of the rank and file in practical medicine. If what I have said is true, I call you to witness how lamentably deficient is the medical teaching which is offered us. On a recent occasion, desiring to get a digest of present conceptions of acidosis and its clinical relations, I turned to the bookshelves, with the following results: In one excellent work on general therapeutics, five volumes, 1914, the word acidosis is not in the index, except as to its relation to diabetes. In another work, covering the same field, three volumes, 1912, the same condition holds. In a widely used single volume on practical therapeutics, 1912, I could not find the word in the indices. In a well-known treatise on dietetics, 1913, the word is indexed only in relation to diabetes. In a deservedly popular "System of Modern Medicine," 1914, a scant two pages is devoted to the subject. Is it not obvious that here lies before us a rich field fallow to our plough?

What must be the present attitude of our Society to best ensure its future development? Since our field of work is practically boundless, we only have to deal with the qualifications of the workers and characterize the expedient limitations of their energies. It is a happy tradition of this Society that the personality of its members making possible more than cordial, even affectionate, intercourse should be

a dominant criterion of fitness in those who would join its ranks. Russell Lowell once said that no long friendship is possible with one from whom we cease to learn.

It is the ideal of happy social intercourse to combine the emotion of fine desire with a sense of intellectual uplift. No more difficult goal could be set up. Herbert Spencer in his autobiography tells of the attempt to bring together in the "X Club" ten men in London who should combine the qualities in question. They never succeeded in enrolling more than nine; but what a table was that round which were gathered Huxley, Tyndall, Hooker, Spencer!

Far be it from me to undervalue the living word and the magnetism of personal contact. But the essence of our civilization consists in the employment of methods by which the influence of the individual is radiated far beyond the bounds of his personal activity.

Many of the very best intellects and richest natures which glorify our profession are, like the "village Hampdens," splendidly fulfilling the duties of their narrow sphere, but exercising no influence beyond it. Now the purpose of a national society is to develop efficiency by the union and co-ordination of local forces. How shall we know what local forces can be profitably incorporated in the national machine? The printing press is the great mechanism of transportation of ideas, and no one without it can send his goods to the world market. Therefore, it appears that a fundamental requirement for fitness in this Society should be scientific publication. But of "making books there is no end," and the good old English custom of prefacing printed contributions with an "apology" might profitably be insisted on in every essay.

A very happy criterion of the fitness for publication of any piece of work is to be found in the query: "Does it

advance medical knowledge?" An unprejudiced negative should automatically suppress the effusion. I do not for a moment intend to imply that our publications should be limited to the exposition of what we fondly believe to be new discoveries in science or art. Clear expositions of known facts, logical theories of actual phenomena, are absolutely necessary to the practical worker. They serve as the basis for the development of knowledge.

Is there any peculiar limitation in the vast field of endeavour open to us which is set by the proper scope of this Society? We may at once admit that by the very ethics of our profession we are denied in most instances the use of the chiefest instrument of scientific research, namely, Experiment.

Though, in a sense, the application of every therapeutic remedy is an experiment, there is a shifting border-line which we dare not cross except at the peril of our trusting patients and danger of rebuke from an outraged conscience.

It is not ours to seek in the laboratory the causes and treatment of disease. Our duties are vastly broader, for they demand exact observation of the phenomena of normal and morbid life, and the effect upon them of every conceivable condition of natural environment and artificial medicament. The enormous output of pathological, bacteriological, immunological, and pharmacological research has supplied us with a therapeutic armamentarium of a complexity which no man understands. Those clinicians whose therapeusis is adopted without question from the teachings of the manufacturing chemist are worse than reactionaries to the days of alchemy. "Judgment is difficult," yet the very essence of the practical physician's right treatment lies in the exercise of good judgment, which is wisdom, which is common-sense. The late W. H. Gaskell, one of the modern masters of

English physiology, wrote: "The race is not to the swift, nor to the strong, but to the wise." These powers are born in us potentially, but can never be put into execution without a sound intellectual training which involves a practical utilization of all the avenues of knowledge and acquaintance with the pitfalls which obstruct progress thereon. This preliminary training is being rapidly and grandly developed under the auspices of our greatest medical organization. Although it is justifiable, nay, imperative, that we daily resort to the use of remedial measures of whose agents and reactions we are profoundly ignorant, nevertheless critical judgment is never to be deleted. The selfsame therapeusis may in the one case be the choice of a master, and in the other of a fool.

The onslaught of bacterial vaccines in our materia medica has put us in the peculiar position of a landsman who undertakes to steer a sailing vessel in a gale of wind; he knows that by a turn of the tiller he may heave to, go forward, or jib; it is only through long training that the sailor's instinct tells him how to carry sail and to steer.

The exigencies of his calling compel the physician constantly to employ agents the nature and action of which he knows but obscurely, and it is demanded of him that he do this with thought, yet not as an experimentalist. We have been reared in the teaching that to "help Nature" should be the guiding motto of the doctor's practice. It is not always realized that Nature holds as dear the pathogenic germ as the higher organism which it invades. As Welch has said, "Nature is neither kind nor cruel." Our intelligence must be the radiant energy which turns pathological into physiological processes. Yet, as philosophical physicians, we must realize that our human endeavours are simply a modern addendum to the eternal forces of evolution.

This is well illustrated by a consideration of the relations of venereal disease to ultimate sexual morality. Promiscuous sexual intercourse, under present social relations, is the principal disseminator of venereal disease. The outcome of such infection for the invaded race is either sterility or immunity. Given time enough—and Nature has no need to hurry—the malefactor is wiped from the face of the earth, or his pathogenic foe turns to a harmless parasite. In the meanwhile the self-protecting instinct of public opinion enters the field; it makes more and more stringent the ban against all that can injure the social order until man's aspirations themselves ordain that sexual desire shall be whipped into the groove leading to eugenic progress of the race. From this point of view evolution uses venereal disease to the perfection of a sexual morality. As a police detective places temptation in the way of a suspect whom he shadows as a test of his inner impulses, so, too, the emotions of man in social congress are the stimuli which strengthen and build up his self-restraint, or which lead him into waters underlaid with quicksands. In evolution there is co-ordinated transformation of the mental and moral with the physical attributes of man.

But now comes an Ehrlich, and by the simple exhibition of a chemical compound abolishes the penalty of the most loathsome case of sex disorder. What must be the ultimate effect of this injection of intelligence into the slowly moving machinery of evolution, what but moral disaster to the race, unless, by compensatory development of instinct and intelligence, mankind can keep pace in his psyche with his mastery of his soma? It follows, as in all human affairs, that increase of power brings increase of responsibility.

Such reflections were inept and inconsequential to a collection of pathologists; but the practising physician is a

mind doctor, and in the end his chief service is that of a comforter.

In conclusion, it appears that the field of endeavour occupied by the American Climatological and Clinical Association is of enormous expanse. No other organization exists which is likely to duplicate the tasks which are properly ours. I am convinced that a scientific society, like a living body, is greatly endangered by redundant, inactive tissue. I believe it would be to the best interests of the Association were its public opinion to insist that every member should be and continue to be a producer for the general welfare. It seems to me that the energies of the Society might advantageously be directed by the appointment each year of special committees for the consideration of definite subjects, such as "The alleged radio-active properties of natural waters"; "The relation of the physical and chemical properties of the air in ventilation"; "The relations of immune sera and vaccines in the treatment of bacterial infections"; "The relative value of sensitized and non-sensitized bacteria in treatment and prophylaxis"; and other themes in which we all have an interest, and with which many have a working knowledge. Even if such a committee report were not founded on personal research, themes of such moment might be chosen that a worthy compilation of existing knowledge would be of great value. Such a committee report should be made the basis of a symposium which would elicit the combined experience and judgment of the Association. The main thought is that our success as an organization depends upon the active co-operation of every member; granting this result, none can doubt the maintenance of the high traditions of this Society, nor that it will continue as a noble monument to the achievements of American Medicine.

THE PHYSICIAN AND THE WEATHER BUREAU.[1]

By FORD A. CARPENTER, LL.D.

U.S. WEATHER BUREAU, LOS ANGELES.

Introduction.

The relation between climate and health has been recognized for at least 2,000 years. The very existence of the American Climatological and Clinical Association is due to this significant relation.

Hippocrates, the Father of Medicine, was the first writer on medical climatology. About 600 years before Christ he wrote a strangely interesting and powerful book on "Airs, Waters, and Places."[2] "Such a title immediately suggests climatology," says Dr. De Lancey Rochester, in a paper read before this Association three years ago, entitled "Climatology as Practised by Hippocrates." He also states that this was one of the genuine works of Hippocrates the Great.

Hippocrates, in his introduction to this work, says: "Whoever wishes to investigate medicine properly should proceed thus: In the first place consider the seasons of the year, and what effects each of them produces (for they are not all alike, but differ much from themselves in regard

[1] Published by permission of the Secretary of Agriculture.

[2] Transactions of the American Climatological Association, 1912, xxviii.

to their changes). Then the winds, the hot and the cold, especially such as are common to both countries, and then such as are peculiar to each locality. We must also consider the qualities of the waters, for as they differ from one another in taste and weight, so also do they differ much in their qualities. In the same manner, when one comes into a city to which he is a stranger, he ought to consider its situation, how it lies as to the winds and rising of the sun; for its influence is not the same, whether it lies to the north or to the south, to the rising or to the setting sun."

How the Physician may utilize the Weather Bureau.

As Dr. Charles F. Marvin, the Chief of the United States Weather Bureau, said in a recent address: "The functions of the Weather Bureau of the United States Department of Agriculture are so many, so widespread is its scope, that it may be said to encompass practically all fields of life and work." From the very extended nature of the work of the Bureau it seems opportune that the medical profession be shown some of the ways in which this service may aid them.

The object of this paper, then, is to suggest means by which the medical profession may utilize the vast storehouses of climatic data accessible at Washington and at all Weather Bureau stations; to prove the readiness of the personnel of the Bureau to assist the physicians; to present some applications of these data from a layman's viewpoint; and to offer an account of some intensive climatic studies which have been prosecuted by the writer during the past few years.

Everyone is familiar with the ordinary health resort advertising literature, a perusal of which gives one the impression that ideal weather conditions only prevail in that locality. Now physicians want facts. They prefer to make

their own deductions. They do not want idealizations. The Weather Bureau presents these facts. The question is, How may these facts be obtained?

The Source of Meteorological Data in the United States.

The source of weather data is from the regular stations of the Bureau, and from the special or co-operative stations. There are about 200 of the regular stations, and over 4,000 of the special stations scattered over the United States. The

Approximate Number of Co-operative Stations of the United States Weather Bureau located in the various States of the Union.

(Total number in the United States, 4,083.)

Alabama	61	Maryland and Delaware	46	Ohio	117
Arizona	80	Michigan	125	Oklahoma	84
Arkansas	47	Mississippi	71	Oregon	137
California	315	Minnesota	82	Pennsylvania	115
Colorado	126	Missouri	78	South Carolina	53
Florida	75	Montana	111	South Dakota	89
Georgia	68	Nebraska	133	Tennessee	69
Idaho	101	Nevada	50	Texas	206
Illinois	96	New England States	85	Utah	139
Indiana	70	New Jersey	45	Virginia	56
Iowa	106	New Mexico	160	Washington	136
Kansas	134	New York	111	West Virginia	58
Kentucky	47	North Carolina	87	Wisconsin	94
Louisiana	62	North Dakota	81	Wyoming	77

Note.—The co-operative stations enumerated in this list are taken from the "Climatological Data from the United States by Sections," March, 1915, vol. ii, No. 3. As all such stations report precipitation, and some stations report this element only, the totals were prepared by considering the precipitation stations.

geographical distribution of these regular and co-operative stations may be noted from the above table and fig. 1 on p. 14.

The regular stations of the Bureau average one to 15,000 square miles of territory. There is no limit to the special stations except the securing of congressional appropriation for purchase of instruments, and the securing of a satisfactory

observer proffering his services in a locality where observations are desired. These special stations, however, must be twenty miles apart. In number of stations, of all kinds, California takes the lead, having a total of 511 meteorological stations of all classes.

Located at each of the Weather Bureau stations are one

WEATHER BUREAU STATIONS IN THE UNITED STATES.

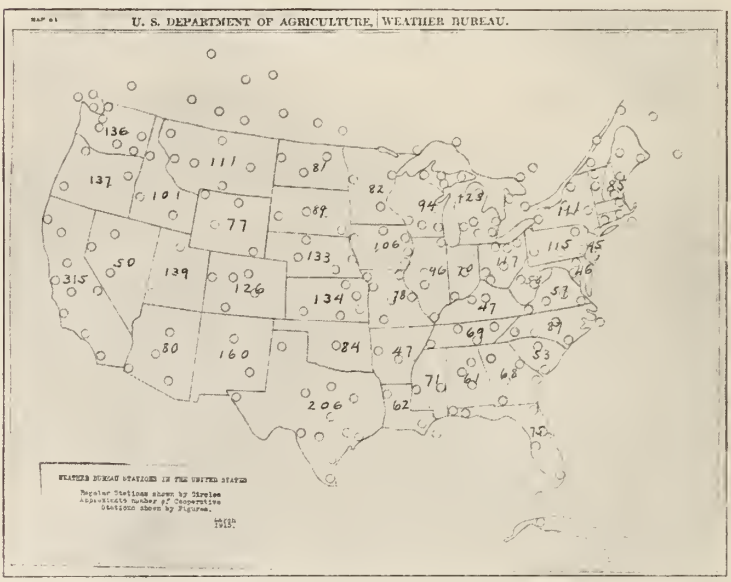


FIG. 1.—The geographical position of regular Weather Bureau stations is shown by circles; the approximate number of co-operative stations is designated by figures.

or more trained observers; such stations are equipped with barometers, thermometers, wind-vanes, rain and snow gauges, and anemometers, and nearly all have sunshine recorders, barographs, thermographs, and other apparatus which make a continuous automatic record of the local weather conditions.

How the Weather Bureau may be utilized.

The prime object of the regular stations is to make simultaneous meteorological observations from which the twice-daily weather map is prepared and weather forecasts are deduced. The duties of the non-commissioned and unpaid observers comprise daily temperature and precipitation observations, so that from both classes of observations it is possible to construct a satisfactory climatology. So we may say it takes a lot of weather to make a climate.

The regular stations of the Bureau, in addition to being clearing-houses for meteorological information, prepare twice a day complete weather observations, which are telegraphed to Washington and to certain district centres preparing weather maps. Over a hundred such stations publish these daily maps. The weather map may be most useful to the physician who keeps track of the storm movements and gathers first-hand information as to existing and coming weather conditions over the United States. He will soon learn to associate stormy, cloudy, and comparatively warm weather with those atmospheric whirls called "lows," and fair, sparkling, comparatively cool weather with the whirls called "highs."

As to the further application of this weather map: One of the large California citrus fruit growers times the shipment of his orange and lemon cars so that they will have the advantage of the cool and dry weather of the weather map "highs" in their eastern journey. There is no reason why a physician should not do likewise and take advantage of the weather map and route his patients according to the great atmospheric movements, and thus avoid deleterious weather conditions. It ought to be a common occurrence for the physician to consult the Weather Bureau as to the

railroad altitudes and average weather conditions along their lines, so that advantage could be taken of railroad routes having low altitudes, warm winter and cool summer weather.

The Scope and Availability of Climatological Data.

As to the climatological work of the Bureau, we have from the Central Office of the Weather Bureau[1] the following data: "Although the 200 regular observing stations, each representing about 15,000 square miles of territory, furnish sufficient data upon which to base the various forecasts, observations at many intermediate points are necessary before the climatology of the United States can be properly studied. This need has given rise to the establishment of an important and interesting feature of the Weather Bureau in its Climatological Service, which is divided into forty-four local sections, each section, as a rule, covering a single State, and having for its centre a regular observing station. These centres each month collect temperature and precipitation observations from more than 4,500 co-operative and other stations, and publish each a monthly and an annual summary, giving a large amount of climatological data for each month and the year."

It is thus seen that the co-operative observers through long periods of temperature and precipitation observations have enabled the Bureau to perfect extensive records. In addition to publishing monthly climatological data in the *Monthly Weather Review* (the first volume was issued with the first of the year 1873), special contributions in climatology and meteorology are published along with the usual profiles, charts, and tables. Bulletins prepared by the scientific staff are also issued by the Bureau, no fewer than seventy having

[1] "The Weather Bureau, 1915," Washington, Government Printing Office.

been published. One of these bulletins is the incomparable "Climatology of the United States."[1] The eminently practical arrangement and the thoroughness of this volume makes it one to be earnestly recommended to the physicians interested in climatic study. A copy of this book will be found in the station library of any Weather Bureau office. While the local libraries are as complete as circumstances may permit, and form a veritable mine of climatic data, these half a thousand volumes are insignificant when compared with the Central Office library in Washington with its 32,000 titles in meteorology. This is the largest meteorological library in existence. It is available to students of meteorology generally, who either consult it personally or through correspondence. This correspondence feature makes it possible readily to consult rare and ordinarily unavailable books.

Local offices of the Weather Bureau have complete records covering continuous registration of the meteorological elements. An abstract of these records is issued at all stations of the service on the first day of the month following. A copy of one of these summaries follows :—

In addition to placing the available meteorological records at the disposal of the applicant, the Weather Bureau officials throughout the country will be found prompt and courteous in their receipt of applications for data in person or by correspondence. In many instances they will render unexpectedly valuable aid to the physician in giving him clear and concise data perhaps not comprehended by their official routine, yet none the less accurate and satisfactory. From their technical training they are keen observers, and their judgment on climatic matters is free from local prejudice.

[1] Washington: *Bulletin Q*, "Climatology of the United States," Henry, 1906.

United States Department of Agriculture.
WEATHER BUREAU.

C. F. MARVIN, Chief.

MONTHLY METEOROLOGICAL SUMMARY.

LOS ANGELES, CALIFORNIA, FOR MAY, 1915.

Date	Temp. Highest	Temp. Lowest	Temp. Mean	Precipitation (inches)	Character of day	Percentage possible sunshine
1	55	41	43	·03	Cloudy ..	31
2	55	44	50	·54	Cloudy ..	13
3	65	48	56	·00	Pt. Cldy. ..	67
4	61	51	56	·29	Cloudy ..	8
5	61	48	54	·00	Pt. Cldy. ..	37
6	63	52	58	·00	Cloudy ..	13
7	65	54	60	·00	Pt. Cldy. ..	54
8	69	53	61	T.	Pt. Cldy. ..	57
9	68	54	61	·00	Pt. Cldy. ..	62
10	70	55	62	·00	Pt. Cldy. ..	59
11	74	55	64	·00	Clear ..	72
12	74	55	64	·00	Clear ..	80
13	67	55	61	·00	Pt. Cldy. ..	59
14	69	53	61	·00	Clear ..	100
15	82	57	70	·00	Clear ..	100
16	78	57	68	·00	Pt. Cldy. ..	81
17	64	54	59	·02	Cloudy ..	33
18	70	51	60	·00	Clear ..	79
19	70	51	60	·00	Clear ..	99
20	69	51	60	·00	Clear ..	76
21	70	51	60	·00	Pt. Cldy. ..	79
22	69	52	60	·00	Pt. Cldy. ..	69
23	71	51	61	·00	Pt. Cldy. ..	36
24	68	54	61	·00	Pt. Cldy. ..	61
25	69	54	61	·00	Pt. Cldy. ..	68
26	77	54	66	·00	Clear ..	87
27	95	64	80	·00	Clear ..	100
28	85	59	72	·00	Pt. Cldy. ..	91
29	69	59	64	·00	Cloudy ..	9
30	71	58	64	·00	Pt. Cldy. ..	65
31	72	55	64	·00	Pt. Cldy. ..	60

This month since 1877	Year	Mean temp.	Total precipitation
	1877	—	—
	1878	63	0·66
	1879	62	0·24
	1880	65	0·04
	1881	64	0·01
	1882	64	0·63
	1883	64	2·02
	1884	64	0·35
	1885	66	0·05
	1886	65	0·00
	1887	65	0·20
	1888	63	0·02
	1889	63	0·62
	1890	63	0·03
	1891	62	0·31
	1892	62	2·06
	1893	63	0·06
	1894	60	0·20
	1895	64	0·19
	1896	63	0·30
	1897	63	0·10
	1898	60	1·75
	1899	60	0·04
	1900	64	1·81
	1901	60	1·50
	1902	61	0·03
	1903	61	T.
	1904	62	T.
	1905	61	0·95
	1906	60	1·02
	1907	61	0·07
	1908	60	0·25
	1909	60	0·00
	1910	63	0·00
	1911	61	0·02
	1912	63	0·12
	1913	61	0·05
	1914	60	0·43
	1915	62	0·88

Mean highest temp. .. .. .. 69·7
Mean lowest temp. .. .. .. 53·2
Mean temp. for month .. .. .. 61·5
Total inches of precipitation during this month .. .. 0·88

WEATHER.

Number of clear days 9; partly cloudy 16; cloudy 6; on which ·01 inch or more rainfall occured, 4. Mean monthly relative humidity, 74 per cent.

SUNSHINE.

Number hours actual sunshine .. 265·2
Number hours possible .. .. 432·1
Percentage of possible 61

PHENOMENA.

	Dates
Auroras	0
Solar halos	3
Lunar halos	0
Hail	0
Fog, light	10, 11, 12, 23, 31
Fog, dense	23
Thunderstorms	0
Frost, light	0
Frost, heavy	0

SUMMARY

BAROMETER.

Mean 29·91 inches; highest, 30·14 inches, 9th; lowest, 29·66 inches, 28th.

TEMPERATURE.

Highest 95, on 27th; lowest 41, on 1st; greatest daily range 31, on 27th; least 10, on the 4th; normal for this month, 62·5; absolute highest this month for 38 years, 103 (25th, 1896); lowest, 40 (16th, 1883); average daily deficiency of this month as compared with normal, 0·1; accumulated excess since January 1st, 184.

PRECIPITATION.

Total for this month, 0·88; snowfall, 0; greatest precipitation, 24 hours, 0·57, 1st-2nd; normal for this month, 0·48; total from September 1st, 1914, to date, 17·04; normal from September 1st to date, 15·57; excess from September 1st, 1914, to date, 1·47; average seasonal (37 years) 15·55.

WIND.

Prevailing direction, S.W.; total movement, 4,732 miles; average hourly velocity, 6·3 miles; maximum velocity (for 5 minutes), 28 miles per hour, from N.W. on 19th.

NOTE.—"T" indicates trace of precipitation.

FORD A. CARPENTER,
Local Forecaster.

Specimen of the Monthly Meteorological Summary issued at all stations of the United States Weather Bureau, and available for the use of the public. Physicians may be placed on the mailing list upon application to the official in charge of the station from which climatic data is desired.

Some Popular Misconceptions relating to Climate and Health.

Air-pressure does not directly affect Health.

From the multitude of problems recently presented, the one considering the relationship between air-pressure and health appears now to be the most popular. There is considerable misconception regarding this subject, occasioned primarily by confusing effect with cause. The mere change in atmospheric pressure from day to day has nothing whatever to do with one's physical well-being. The recent publicity given to this subject during the last few months is simply a revival of a myth laid a generation ago by Thomas. For example, in a recent public bulletin it is stated that when the barometer drops from 30 to 29 in from one day to the next, the human body is relieved of half a ton of atmospheric pressure, with resulting depression of spirits, &c. In the first place, it is quite unusual for the barometer to drop 1 in. in so short a time, and in the second place, experiments have proved that the influence upon the body of variations in pressure are greatly over-estimated.

Hann,[1] in his handbook of climatology translated by Professor Robert De C. Ward, quotes Thomas, and says that in experiments with pneumatic chambers, pressure changes amounting to 300 mm. [over 11 in. in barometric values] a day have been produced without causing any injurious effects upon the sick persons concerned in these experiments. Although these experiments were made nearly half a century ago the pressure myth has persisted, and even this year has found its way into sober-minded periodicals. If a local example is needed further to demonstrate

[1] N. V. Hann (Thomas: "Beiträge zur allgemeinen Klimatologie," Erlangen, 1872).

the intenability of the pressure theory, we have only to recall that in Los Angeles, for example, the range of the barometer from day to day averages a tenth of an inch of the mercurial column, equalling the variation experienced when riding in an elevator from the ground floor to the top of office buildings. If there was anything in this theory we would have an elevator disease among the elevator conductors, who continually experience excessively rapid fluctuations in bodily pressure.[1]

It is rather the fair weather and decreased humidity which accompany rising barometric pressure that elevate one's spirits, or the cloudy, threatening weather and increased humidity which accompany a falling barometer that affect the human system.

Dr. C. C. Browning[2] notes this when his studies showed that the tubercular patients became pessimistic and some very unreasonable in their demands during days with little or no sunshine, and hopeful of recovery during bright days and moderately low humidity.

Sunshine and Ventilation of more Importance than Relative Humidity.

I believe that the location of the home or office, the exposure of the living- or work-room has much more to do with the health of the occupant than whether or not the relative humidity is much below or above the normal. Exposure to sunshine and ample ventilation are all-important. A friend complained of ill-health, and blamed the humidity

[1] Since the above was written, the author, while on duty at the Signal Corps Aviation School at San Diego, Cal. (as lecturer on certain phases of meteorology applied to aviation), made an ascent of 3,000 ft. on October 20, 1915. Although the aeroplane reached this altitude in a few minutes, the barometer falling from 30·0 to 27·0 in., the decreased bodily pressure was not at all noticeable.

[2] Transactions of the American Climatological Association, 1913, xxix.

of his office room. He said that it was too damp and asked me to investigate. I took my psychrometer and made a series of observations, finding that there was actually less humidity in his private office than out in the street. I studied the situation and eliminated temperature, humidity, air-pressure, &c. Then I turned to air-movement, and found the ventilation very bad. I suggested a simple air-changing system, and my friend regained his health in a few weeks, and was still enjoying his private office when I last heard from him.

Living Quarters and Air-drainage.

Facing houses with relation to natural air-drainage is a most important point in selecting a home. Many a home, otherwise healthful and satisfactory in every other respect, has been abandoned because the architect and owner did not consider the principles laid down by Hippocrates two thousand years ago.

Dr. Estes Nichols, at last year's meeting of this Association, said, in his paper on "Housing and its Relation to Climate":[1] "Can any climate rise above its housing conditions? I offer no theories as to the building of houses, as all theories are apt to be overthrown by each succeeding year. I only offer the one fact that has been so well established, and that is, the vital necessity of plenty of room, as well as plenty of light and air, even to the remotest part of our dwellings."

PROBLEMS FOR THE CLIMATOLOGIST.

Investigation by the Physician.

There are other opportunities for the study of health and its relation to climate, as further TRANSACTIONS of this Association show. In the light of augmented data, a few of them

[1] TRANSACTIONS OF THE AMERICAN CLIMATOLOGICAL ASSOCIATION, XXX, p. 17.

might be, for example, the proper selection of sanatorium sites, sensible temperature (wet-bulb thermometer readings), and infant mortality; acute infectious diseases and sunlight (using the sunshine charts of the Weather Bureau), &c. These are within the scope of the medical profession; the Weather Bureau, however keenly interested, can only assist with its climatic data.

Intensive Studies by the Climatologist.

There is one phase of the relationship of the physician to the Weather Bureau where the balance of proof falls upon the weather service, and that is in the matter of intensive climatic investigation. Primarily, owing to the extensive citrus fruit culture in which the United States Department of Agriculture is deeply interested, some of these studies have been taken up in Southern California during the past year by the local office of the Weather Bureau at Los Angeles. Indirectly the mass of data is of much value to the medical profession.

Southern California is about equal in area to that of Indiana, and within the boundaries of this region may be found all kinds of varieties of healthful weather. This is owing to the mountains, the sea and the desert, as well as the latitude. Scattered over this one portion of California are a dozen stations equipped with thermographs and hygrographs, rain-gauges, &c. An examination of the curves of temperature and relative humidity as automatically traced is a most interesting study. Two sets of these curves are illustrated in fig. 2, p. 23. They show simultaneous tracings of temperature and relative humidity during a week in June. One of these stations, Los Angeles, is within 15 miles of the sea, and shows lower range in temperature and

higher relative humidity; and the other, Riverside, which is 55 miles east towards the desert, illustrates most clearly the influence of the desert in its range in temperature and low humidity, and above all for its remarkably regular oscillations. Such a graphic representation of the

WEEKLY THERMOGRAMS AND HYGROGRAMS.

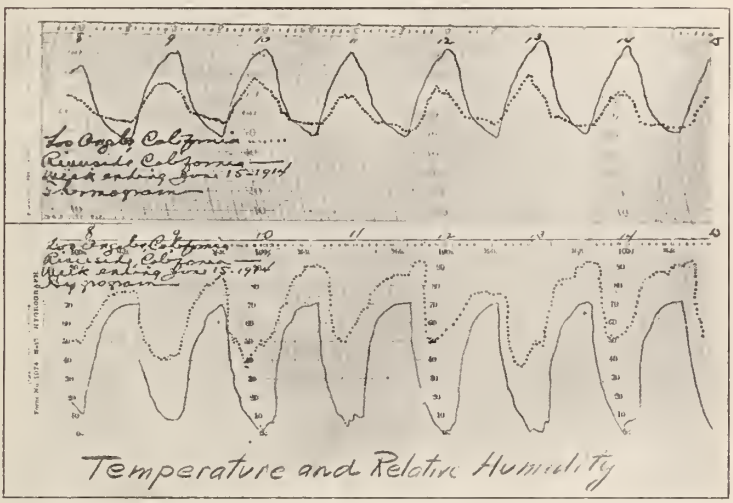


FIG. 2.—Variation in temperature and relative humidity between Los Angeles (15 miles from the sea) and Riverside (40 miles from the sea) for the same week ending June 15, 1914.

diversity of climate within so short a distance would be well-nigh impossible save in Southern California.

Intensive Meteorology to the Aid of Colonization and Health.

For the first time in history climatic surveys are being made to determine land values. This is a practical business-like, commercial application of meteorology to colonization and intensive farming. This climatic survey was begun in

Intensive Climatic Charts of Temperature and Humidity.

Nearly due south of the city of Los Angeles lies the rugged sea coast on which is located the old Spanish grant of Los Palos Verdes. In June, 1914, was begun a series of automatic meteorological records comprising temperature, humidity, sunshine, wind and rainfall. These charts show the variation in temperature and humidity, with elevation and proximity to the ocean.

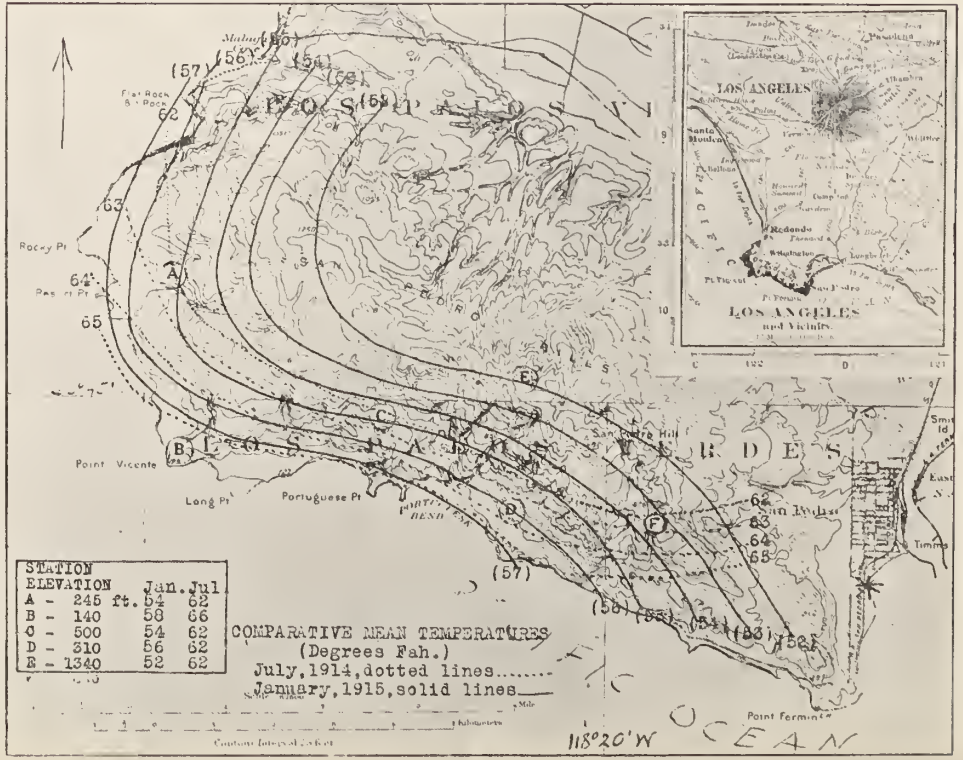


Fig. 3.—Mean temperatures for January and July at Los Palos Verdes, near Los Angeles.

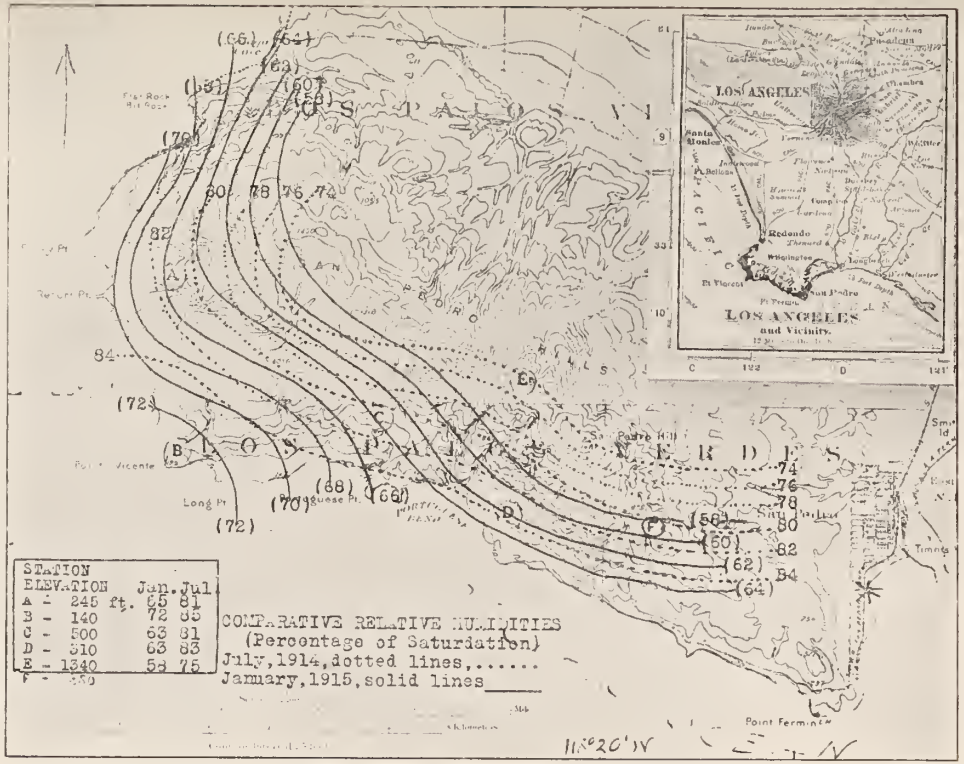


FIG. 4.—Mean relative humidities for January and July at Los Palos Verdes, near Los Angeles.

June, 1914, on the old Spanish grant of 16,000 acres, the Palos Verdes, which is just south of Los Angeles, and between San Pedro (Los Angeles harbour) on the east and Redondo beach on the west. Six complete sets of meteorological instruments, comprising standard thermographs, hygrographs, rain-gauges, anemometers, and a photographic sunshine recorder, were erected at various elevations from 100 ft. to 1,300 ft. above the sea, and from the sea-shore to several miles inland. The readings of these instruments have been plotted, and comparative charts of temperature and humidity for January and July are shown in figs. 3 and 4, facing pp. 24, 25. With such intensive study it is believed possible to chart the climate for every ten-acre plot. The climatic charts will show without error not only proper location of dwellings with relation to air-drainage, temperature, humidity, &c., but will give positive information as to soil-drainage, and all of this in addition to the all-important agricultural and horticultural features.

What the Weather Bureau should do from a Physician's View-point.

In closing this paper, I feel that a physician friend who has long practised medicine in one of the towns in California has clearly stated some of the ways the physician may properly claim the assistance of the Weather Bureau. This eminent physician says: "I believe the field has been but little explored. We physicians are guilty of many things; among them is guessing. If the United States Weather Bureau will prove some of this guessing true or false it will add one more good thing to its past fine record. I think that the time is near at hand when our health officers will be required to be specialists. They will then have the time,

the special training, and other equipment to go into the subject in conjunction with your Bureau and give us some scientific findings. I think the State should be plotted, showing the real atmospheric conditions of all localities, and showing the influence upon the functions of the body. Whether the "cold east wind" of Boston is accountable for all the vile things charged to it or not nobody really knows, but it should be investigated. That altitude, temperature, humidity, prevailing and unusual winds have much effect upon many individuals there is no doubt. The nasal and pulmonary mucous membranes are constantly affected by atmospheric conditions. Locally, I have observed semi-chronic bronchial coughs that do not yield readily in the city will often clear up in a few hours in the mountains and foot-hills east of the city. A congested nasal mucous membrane that will kick up a rumpus much of the time in the mountains will disappear promptly at a lower elevation near the coast. Some cases of bronchial asthma that are incurable in the business section of the city will be very comfortable a short distance out of town and in a higher, drier location. Two hot dry days last month have been charged up with some rather serious pulmonary conditions in aged people. These are a few instances of hundreds coming to my mind that I have observed in my years of practice. The subject should be worked out by a union of effort of the Weather Bureau and the medical profession."

DISCUSSION.

Dr. HENRY SEWALL (Denver, Colo.): There are several things, if I may take up time in the discussion, that are not clear to me. From the first chart shown, I could scarcely grasp how it is that the intense heat is produced from the proximity of high pressure to low pressure,

or of pressures of unusual intensity side by side. It is so clear to the expert that he did not think it necessary to explain it. It surprised me to find in arid regions the tremendous humidity reported—95 per cent. —in a region which publishes a mean humidity of something like 5, 10, or 15. I do not quite grasp the situation. One can see that if the air is chilled sufficiently a relative humidity will ascend proportionately, but I did not suppose that the chill in the air in those regions was sufficient to produce such a condensation. I have wondered whether those high humidities were due to the transportation of moisture from outside. These things occur to the amateur in reading the meteorological data which is so familiar to the meteorologist.

Dr. CHAS. C. BROWNING (Los Angeles) : I think we are indebted to Dr. Carpenter for bringing this paper before our Society. It brings to us a different view-point from which we who are physicians are accustomed to view weather conditions—that of scientific observations accurately recorded. I think each of us come to rely greatly on impressions regarding weather conditions, changes and influence on our patients, rather than on records. That these impressions are of value, I do not doubt, but they are of greater value when founded on, or checked by accurate observations.

This is illustrated by statements frequently heard that the climate is changing, and persons give their experience in proof of this fact. That extremes do occur is borne out by the records—one notably in 1913, when trees, cacti, &c., known to have been more than 100 years of age were frozen, but the charts presented to us to-day shows that the average is not changing perceptibly—not enough to be noticeable within the life of a man.

I have availed myself of some of the privileges accorded by our local Weather Bureau and with advantage. I was surprised when I made my first application to find the cordial welcome I received. I had not expected that the officers of the Bureau would appreciate the interest a physician might have, but the suggestions made in this paper indicate that they are alive to this phase of our work and should be an incentive for closer co-operation on our part.

Dr. JOHN A. LICHTY (Pittsburgh, Pa.) : I think this class of papers exceedingly valuable to us because it brings a view-point based on scientific observation which we do not get otherwise.

I would like to ask Mr. Carpenter whether in his opinion weather conditions in any community change very much from time to time? So often we have old residents say that the winters are "not so cold" as they used to be, "the snow not so deep," &c.

A MEMBER : I would like to ask a question with reference to the change of the weather from year to year. The speaker states that these conditions maintain the same average. I suppose we all have had the same experience—that in our boyhood days the winters were longer, the snows deeper, the weather colder, &c., than it is now, just as the

mountains seemed higher than now. I was making a trip through Salt Lake basin the other day and was speaking with one or two men at Salt Lake City. They told me that when the Mormons first arrived there they had terrific winters and snows, which they do not have at all now. I have heard several times that in irrigated valleys the weather conditions change. I wonder whether this general statement included also these changes which have been taking place through irrigated regions.

Dr. W. Jarvis Barlow: This is such an important subject to our Association may I ask that Mr. Carpenter explain the relationship between extremes of temperature and extremes of relative humidity, as in the illustration of relative values at Riverside and Los Angeles?

Mr. Carpenter: Whether it be desert or seashore the amount of moisture in the atmosphere rapidly increases with the growth of the day, but with the augmented temperature the moisture-content of the air becomes greater and the relative humidity decreases. In the lantern-slide shown it will be observed that the time of highest temperature also marks the time of the lowest relative humidity at both Los Angeles and Riverside. Until the general introduction of recording apparatus depicting relative humidity throughout the twenty-four hours it was popularly supposed that the desert was always dry and that the air in such regions showed little diurnal change. This error could readily have been detected if observations had been made of the dew, which we know is frequently and abundantly prevalent in arid countries. In the illustration given of the thermograms and hygrograms of Los Angeles and Riverside it will be noticed that the maxima of temperature and minima of relative humidity occur practically simultaneously at both of these stations. The practical value of automatic trace-sheets in intensive climatic study was forcibly brought to my attention not long ago when a visitor applied for comparative data of temperature and humidity between Los Angeles and points several hundred miles distant, to which latter locality he had decided to emigrate on account of the health of his wife. Before incurring the considerable expense of moving I suggested that he wait until trace-sheets could be secured from the localities he had selected. These sheets were compared with local values and also with neighbouring desert towns, with the result that within a few hours' travel out of this city he found exactly the climate he needed and one superior to those he thought would be satisfactory.

Dr. Sewall (referring to weather map values): In winter, is not the area of high barometer frequently an area of intense cold?

Mr. Carpenter: It is so because the area of low temperatures (a cold-wave) closely follows such a field of high barometer. It is the relationship of these waves of atmospheric pressure that give us our cold and our warm weather. Sometimes an area of high pressure will cover the greater part of the United States. At such times the weather

is fair, only on the outer edges of this field is the weather cloudy or stormy. The high as well as the low areas move from west to east making a procession of crests and hollows of atmospheric waves which march spirally around the pole in a generous circle.

Dr. SEWALL: Does the linear distance between the area of high pressure and low pressure—one or a thousand miles—influence your temperature along that line?

Mr. CARPENTER: It does, for the reason that the steepness of the gradient determines the relative degree of temperature. Familiar examples of this may be observed in the weather maps during "norther" conditions over the southern portion of the United States. In Southern California high temperatures are always the result of an area of high barometric pressure acting in conjunction with a low area, the former over the north Pacific coast (or eastward occupying the plateau region), and the latter over the valley of the Colorado river.

Dr. J. A. LICHTY: Does an examination of the extensive climatological data collected by the Weather Bureau show that the climate is changing?

Mr. CARPENTER: We have in the United States several temperature and rainfall records of 100 years. On the Pacific coast, at San Diego, we have a splendid meteorological record of sixty-six years of observations conducted by the various branches of the Government. We find in those records that the curves of variation in temperature and rainfall show no progressive change either towards increased precipitation or aridity or gradually increasing or decreasing temperature. A careful study of these records does not reveal either a progressive or a periodic change in the climates of the United States. If we study the records of harvest, existence of forests, rivers, lakes, &c., we find that there has been no appreciable change in the climate of the earth since the dawn of history, say, ten thousand years.

THE EFFECT OF CHANGES IN ATMOSPHERIC CONDITIONS UPON THE UPPER RESPIRATORY TRACT.[1]

By JAMES ALEXANDER MILLER, A.M., M.D.
AND
GERHARD HUTCHISON COCKS, M.D., F.A.C.S.
NEW YORK.

[READ BY TITLE.]

While the importance of fresh air in the preservation of health and the value of open air for the treatment of the various forms of disease are widely appreciated, our knowledge of the fundamental principles upon which the action of fresh air upon the body in health and disease depends is inadequate and to a considerable extent unsatisfactory.

Principles of Ventilation.—Under ordinary conditions of life, when so much of our time is spent indoors, we are dependent for fresh air upon the sources of ventilation, and yet we find that at the present time no inconsiderable amount of confusion as to what constitutes proper ventilation exists in the minds of engineers and hygienists alike. This appears to be no fault of the mechanics involved, as the engineers confidently assert their ability to adequately meet any standards that may be generally adopted as representing

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[1] Conducted under the auspices of the New York State Commission on Ventilation.

good ventilation, and that all that is needed are reliable standards.

Progress has been made to this extent, that we can confidently discard the chemical constituents of the air as of no great importance. It has been demonstrated that the amount of oxygen or the proportion of CO_2 may vary between very wide limits without producing physiological effects. Moreover, the effects of poor ventilation can no longer be explained by the presence of volatile organic poisons in the air, such as was first suggested by Brown-Sequard [1] and more recently supported by the experiments of Rosenau [2], for later investigations appear to disapprove these contentions.

The tendency at present appears to be to explain the physiological effects of air on the human body in terms of the physical influence exerted, the principal factors being temperature, humidity, and air movement.

Hermans [3], in 1883, was the first to suggest that the bad effects of poor ventilation are due primarily to the inability of the body to cool itself because of the increased temperature and moisture of the surrounding air. Since that time many other observers, notably Rubner [4], Flugge [5], Haldane [6], and more recently Leonard Hill [7] and his colleagues, have contributed very largely to our knowledge of the action of various conditions of the atmosphere upon the human body, both in chamber experiments on man and animals, and also by numerous clinical observations under various conditions.

While the general principles appear to be established, there is still a considerable lack of uniformity in the ideas expressed as to just what variations in the atmosphere are most harmful and which most beneficial, and particularly as to the methods by which conditions within doors, whether

in houses, workshops, public halls, &c., may be properly controlled in order to maintain conditions of comfort and health. Elaborate systems of ventilation installed at great cost have been very generally condemned, and the increasing popularity of the fresh-air idea has led to a very considerable reaction toward the natural or open-window method of ventilation. Whether this is justified or not is far from certain.

When we turn to the problem of the treatment of disease, particularly respiratory diseases, both acute and chronic, we find, of course, the open-air method generally accepted and widely adopted, but here, too, we may note the lack of accurate knowledge of certain principles which would appear fundamental. If the important factors in proper ventilation are the temperature, humidity and movement of air, they probably are no less so in the application of open air to the treatment of disease.

It is needless to observe that open air varies from day to day, and even from hour to hour, in respect to these three conditions, but particularly is it true that between different localities these variations are extremely great; yet we have very little accurate information as to just how these factors influence patients with respiratory diseases. Still less do we know what conditions may be considered ideal. It is also logical to presume with the marked variations in the types of these diseases, and also in the air effects upon the respiratory organs, as well as upon the body as a whole, that the reaction to the same air environment will differ materially in different patients.

In our acceptance of the value of open air we tend to discriminate too little as to just what conditions of open air exist, and also to consider little, if at all, the conditions we like best to obtain for our varying types of patients.

If our knowledge is somewhat inexact as to what influence

these conditions have upon the body as a whole, it is distinctly more so when we study their exact effect upon the respiratory organs themselves. That this effect is considerable is generally acknowledged, and yet there is lack of carefully controlled observations and very marked difference of opinion as to the effects of changes in the air and their method of action upon the respiratory tract.

General Physiological Considerations.—It may not be amiss to rapidly review certain fundamental physiological considerations.

The internal temperature of the healthy man is fairly constant, varying not more than 1° to 2° F. in the Tropics or in the Arctic regions. Moreover, the temperature of man is more readily adapted to extreme heat and cold than that of any other animal. This capacity for heat regulation depends largely upon the vaso-motor system and the sweat glands. The production of internal heat, being a result of metabolism, varies greatly with the activity of the various organs of the body, and the ability to maintain the constant temperature consequently depends largely upon the possibility of adequate heat elimination. This elimination of heat takes place to a very considerable extent from the skin, the heat being lost by radiation, conduction, and evaporation. Through the vaso-motor system, the skin responds promptly to variation in external temperature by the contraction or dilatation of the superficial blood-vessels. The amount of heat lost from the surface of the body by radiation and conduction depends largely upon the temperature of the surrounding air, the amount of heat lost from evaporation upon the humidity. Inasmuch as the humidity of the air immediately about the body is rapidly increased by the escaping moisture, the amount of air movement by modifying this "aerial envelope" very materially affects

the heat lost by evaporation. Also, inasmuch as moist air is a far better conductor of heat than dry, the heat lost by conduction is materially affected both by the humidity and air movement. The character and amount of clothing as well as of muscular activity are additional important factors.

Haldane [6], in observation upon miners, found that during rest the rectal temperature did not show abnormal increase until the wet bulb thermometer registered 88° F. But if the wet bulb temperature exceeded this by even one degree a very marked rise of rectal temperature occurred, and further increase caused a still more rapid rise of rectal temperature. In moving air a higher wet bulb temperature (93° F.) could be borne without rise of rectal temperature.

During muscular work in still air the limit was much lower, being 78° F.; during leisurely climbing, but if the air was moving, 85° F. could be borne without rise of body temperature.

The chief criticism of available data concerning the effect of atmospheric temperature upon the body is that they are very largely based upon extreme conditions which do not obtain under ordinary methods of living.

Effect of Changing Temperatures.—Change of atmospheric temperature in itself is claimed to have effects which, within reasonable limits, appear beneficial, a sudden change from warm to cold toning up the vascular system; and when frequently repeated it is assumed to produce a healthy exercise of the muscular tissues involved so that the power of prompt accommodation to change of temperature is increased and the individual becomes less liable to the ill-effects of either high or low temperature.

Walter James [8] has suggested this as an important point to be observed in ventilation. He believes that an

even temperature lowers the tone of the vaso-motor system by lack of its proper exercise; this exercise he thinks results from the stimuli of changing temperatures.

Jonathan Wright has also expressed to us verbally a similar notion concerning the effect of changes taking place in the nasal mucous membranes as a result of temperatures varying within reasonable limits. We are not aware of any definite proof to substantiate these rather plausible ideas.

Effect of Cold.—The physiological lower limit of atmospheric temperature appears to be less accurately established than the upper. Blood temperature of 68° F. is incompatible with life, but this has only been noted after very long and severe exposure to low temperatures. Just where the physiological advantages of cold temperature pass over into the disadvantages of pathological change has not been accurately determined, excepting that it appears to be extremely variable in different conditions of age, race, sex, and of health and disease.

Exposure to cold has been very generally accepted as a frequent direct or indirect cause of a large number of diseases, particularly those of the respiratory tract. Since the development of modern bacteriology, however, a great deal of doubt has been cast upon the truth of this general belief, but the widespread prevalence of the notion that there exists a certain danger attending exposure to cold should not be too lightly dismissed, and a close study of this problem is needed.

Some experimental evidence exists to show that not only upon the respiratory tract is the influence of cold important, but also in rheumatism and allied conditions, neuritis, neuralgia and myalgia; in nephritis also, and particularly in the interesting condition of paroxysmal hæmoglobinuria. There is also some experimental evidence to support the idea

that resistance to infection is influenced by exposure to cold. Lode [9], Fischel [10], and Durck [11] experimented with rabbits and guinea-pigs. After shaving the animals they were chilled in various ways, and the great majority of them are claimed to have either succumbed to auto-infection or to have proved far more susceptible to inoculations of various pathogenic bacteria than control animals.

Chodounsky [12], on the other hand, experimented with unshaved animals and obtained entirely negative results. He found the chilled animals no more susceptible than the controls.

As an indirect cause of disease, numerous observers have shown that vaso-motor contraction of the skin vessels due to cold is accompanied by reflex dilatation of vessels in other portions of the body, the resulting congestion of certain tissues or organs being held accountable for many of the so-called "cold" diseases.

Rossbach [13] and Aschenbrandt, after opening the trachea of a cat, applied warm compresses to the belly of the animal, followed by ice compresses half a minute later. After applications of the ice they observed vascular spasm of the tracheal vessels with pallor, lasting one to two minutes. This was followed by a gradual vascular dilatation shown by redness, and finally, after five or ten minutes, by a dark purplish colour, accompanied by marked increased secretion of mucus.

Winter [14] claims that the changes in the tracheal mucous membranes are the same as those occurring in the development of inflammation, and therefore represent true catarrhal inflammation. He thinks that a predisposition to catarrhal attacks of this nature follows severe illnesses of childhood when the affected vessels were relaxed without subsequently regaining their tone. Likewise severe muscular

exertion in hot or vitiated air, he claims, leads to active dilatation of the vessels of the respiratory mucous membranes, and if there now follows a sudden exposure to cold he thinks that a condition predisposing to catarrhal inflammation is produced.

Chodounsky [12], in criticizing the results of Rossbach and Aschenbrandt, pertinently remarks that in the treatment of acute abdominal conditions ice applications are frequently employed, and that this procedure has never been blamed for any illness, catarrhal or otherwise. He also states that there is no proof that hyperæmia in itself is in any way detrimental, citing Bier and his results with both active and passive hyperæmia for therapeutic purposes.

Meuzer [15] thinks that the observations of Rossbach and Aschenbrandt are of little value because the trachea was opened and so exposed directly to the outside air. He believes that by warming and moistening the cold air the nasal mucous membranes become dry and anæmic and the blood in its vessels more viscid, resulting in lessened phagocytosis, and that resistance to bacterial invasion is thereby lowered.

Kuhn [16] agrees that long exposure to cold produces dryness of the mucous membranes and so predisposes to cold. He thinks, however, that a short exposure may protect from infection if it is followed by reaction characterized by hyperæmia of the skin and mucous membranes.

Kisskalt [17] believes that this reflex hyperæmia following exposure to cold predisposes to infection, in spite of the fact that Bier uses passive hyperæmia as an aid in overcoming infection. He thinks that passive hyperæmia is protective by increased alkalinity of the blood, but that active hyperæmia predisposes to infection because blood alkalinity is diminished. We can find no proof of this assumption.

Leonard Hill [7], by direct observation with the nasal

speculum, observed that the mucous membranes of the nose became swollen and red in warm, moist air, and that there is a marked increase of secretion. On passing from the warm to cold air, he found that the mucous membrane became paler, but still remained swollen. He believes that this condition predisposes to disease for the reason that the defensive mechanism of the blood, the immunization properties of the plasma, the cleansing action of the cilia, and the phagocytic action of the white blood cells are all diminished by the cold, while the pathogenic bacteria find a suitable nidus for their growth in the secretion of the swollen mucous membranes. He found that keeping the air in motion in warm rooms very materially interfered with the hyperæmic effect, and also believes that in this way the massiveness of direct infection is reduced. He also observed that warm, dry air produced less swelling and secretion than warm, moist air.

Chodounsky [12], in an excellent monograph, contends that respiratory diseases are entirely a matter of infection and that exposure to cold and variation in atmospheric conditions generally have no effect. He conducted very numerous experiments on himself, and also has collected a large mass of statistics in which he attributes the variations in the incidence of the respiratory diseases at different seasons to other factors rather than the changes of temperature.

Many observers hold that respiratory diseases are due to infection from dormant bacteria which remain latent in the respiratory tract, and that disease only occurs when conditions of atmospheric air are such as to render the mucous membranes susceptible to the access of bacteria. Chodounsky has combated this theory both by animal experiments and experiments on himself. For example, during the month of February, while suffering from chronic bronchitis, and

after showing that his respiratory tract contained a number of pathogenic organisms, on six different occasions he exposed his naked body to draughts after a warm bath without drying himself, at a temperature of 0° to 2° C. No infection followed any of his experiments.

Another interesting hypothesis as to the relation of cold to disease is based upon chemical reactions.

Esch [18] believed that during the winter, when living is largely indoors and unhygienic, the blood contains an excess of excretory products, and that during exposure to cold large quantities of this vitiated blood are forced into the internal organs tending to cause disease.

Nebelthau [19], in experimenting with dogs after severe chilling, was able to demonstrate hæmoglobinæmia, but not hæmoglobinuria. These observations are interesting in connection with what we know of the action of cold in paroxysmal hæmoglobinuria.

When we attempt to sum up the results of these various observations and opinions, we find it difficult to arrive at a satisfactory conclusion as to the exact place of atmospheric changes of temperature in the production of respiratory disease. For this reason we thought it worth while to attempt to add something to our knowledge of this subject by direct observations upon the respiratory tract under conditions which could be absolutely controlled as to temperature, humidity, and air movement.

In this work we were ably assisted by Dr. Robert E. Buckley, who helped in making the examinations of the subjects.

The Commission placed at our disposal two adjoining rooms, approximately 10 ft. by 14 ft. each. These rooms were so arranged that any desired degree of temperature and humidity could be secured and recorded.

Room A, the Control Room, is fitted with white-tiled walls, and with machinery for controlling atmospheric conditions in both rooms, viz., fans, air-washer, heating stacks, steam lines and ducts.

Room B, the Observation Room, is also fitted with white-tiled walls covered with cork-board, cement, and impervious paint. The two rooms are adjacent with a window in the common wall.

The temperature and humidity conditions in Room B were recorded continuously by a Bristol recording wet and dry bulb thermometer. In Room A the records were taken by means of a sling psychrometer.

The terms used in defining the conditions of temperature employed are as follows :—

	Fahr.	Relative humidity
Normal	... 68° ...	... 50 per cent.
Cold	... 50° ...	... 50 ,,
Hot dry	.. 80° ...	... 20-30 ,,
Hot medium ..	... 80-86° ...	... 50 ,,
Hot moist	... 80-86° ...	... 80 ,,

As it seemed desirable to substantiate the results of the clinical examinations of the nasal passages in the various experiments by a more exact method, we made Glatzel mirror records in practically all experiments, except those made in the hot, moist room (86° F., 80 per cent. relative humidity). The Glatzel mirror proved very useful for estimating qualitatively the amount of nasal obstruction caused by changes in atmospheric conditions.

Outline of Experiments.

(1) *Series A.*—Examination of 131 cases changing from one atmospheric condition to another.

(2) *Series B. Studies in Air Movement.*—Examination

of seventy-seven cases changing from one atmospheric condition to another with fans blowing directly on the face.

(3) *Series C. Skin Reflexes.*—Examination of fifty-seven cases with body in one room and head in another room having a different temperature.

(4) *Series D. Skin Reflexes.*—Examination of forty-two cases with the body exposed to one temperature and the feet immersed in a water-bath of a markedly different temperature.

(5) *Series E. Studies of Workers exposed to Dry Heat.*—Examination of forty-five cases of workers in dry heat (firemen, stationary engineers, boiler-makers, &c.) on going from one temperature to another.

(6) *Series F. Studies of Outdoor Workers.*—Examination of fifty-eight cases of outdoor workers (truckmen, drivers, &c.) on going from one temperature to another.

(7) *Series G 1. Studies on Cases of Atrophic Rhinitis corresponding to Workers in Moist Heat.*—Thirty-nine observations on cases of atrophic rhinitis on going from a hot, dry room to a cold room, and *vice versa*.

It was found that a large proportion of workers in hot, moist rooms (steam laundries) suffer from atrophic rhinitis. Owing to the difficulty of securing employees of laundries who could be induced to give the time required for the experiments, we were forced to substitute other subjects for this group. Cases of atrophic rhinitis were therefore selected elsewhere, and used in place of actual laundry workers.

(8) *Series G 2. Examination of Forty-six Laundry Workers* while on duty in two steam laundries in New York City.

(9) Experimental observations on the larynx and trachea of dogs subjected to peripheral stimulation by means of heat and cold.

The subjects chosen for the first set of experiments were

selected from young male college students, between the ages of 18 and 23 years, on whom a careful study of the nasal passages had previously been made. At first all subjects with distinct nasal abnormalities, such as septal deflections and hypertrophic rhinitis, were excluded. It was found, as was to be expected, that very few of the men presented absolutely normal nasal passages. As experience demonstrated that these abnormalities did not materially affect the reactions obtained, such cases were later included in the majority of our observations.

The first studies were made on subjects changing from one atmospheric condition to another. We shall describe the method followed in Series A, Test 1, which is typical of the plan pursued in all experiments.

We worked four hours daily, a two-hour period, from 9 to 11 a.m., and two hours in the afternoon, from 4 to 6 p.m. In Test 1, where we studied the effect of the change from the normal room to a hot room with medium humidity, the subjects for the morning experiments reported at 8 a.m. and were placed in the normal room, where they remained for from one to two hours. At the end of this time each subject was examined in the normal room, a careful record of the examination made, and a Glatzel plate record taken. The subjects were then placed in the hot experimental room, examined immediately, and a Glatzel plate record taken at the time when the greatest change was observed clinically. If no change was observed the Glatzel drawing was taken at the end of the session. During the morning experiments the subjects were allowed to remain in the second experimental chamber (in Test 1, the hot room) for from half to one hour. In the afternoon session the effect of the second atmospheric chamber (the hot room) was studied for one to two hours.

Summary of Results of Series A.

In general, the experiments of this series show that on passing from a normal or cold room into a hot room there results, usually, an increase in colour, moisture, and size of the inferior turbinates and nasal mucosa. Conversely, on going from a normal or hot room to a cold, there is a decrease in colour, moisture, and size of the inferior turbinates in a large percentage of the cases.

These changes are shown schematically in Chart 1. The reactions are expressed in percentages of the total number of nostrils examined.

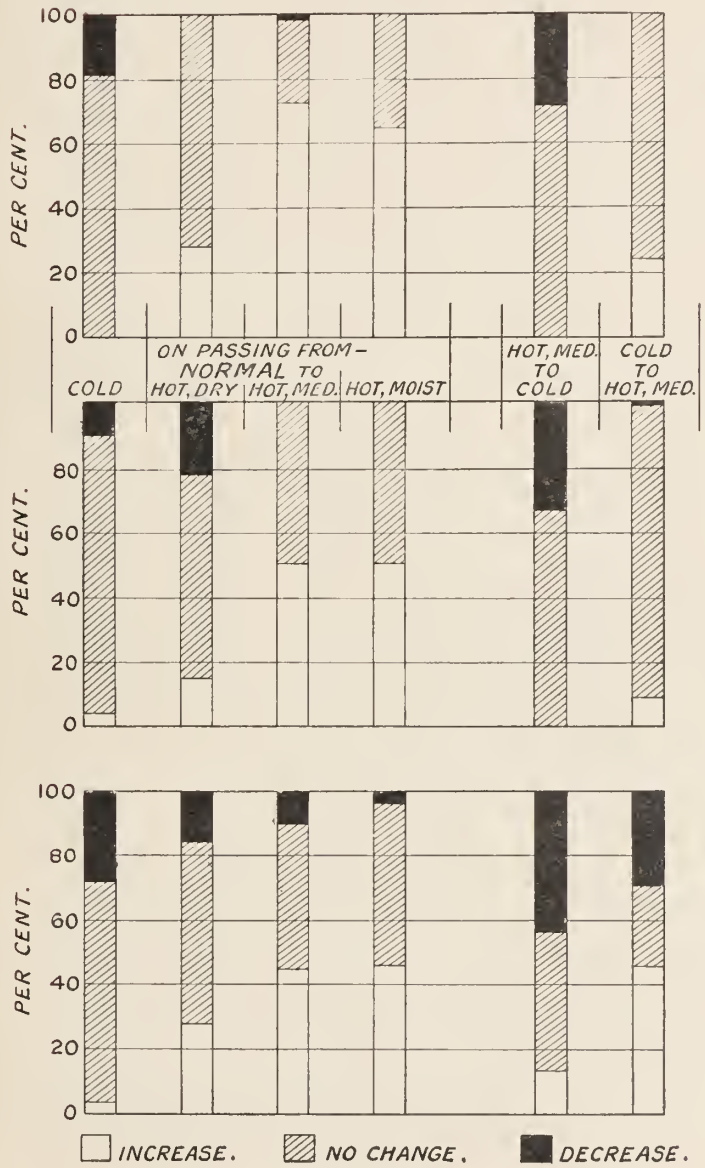
The first column on the left of the chart represents the reactions observed on going from the normal chamber into the cold chamber. Here we note that 28 per cent. of the nostrils exhibited a decrease of the inferior turbinates, 4 per cent. an increase of the inferior turbinates; in 10 per cent. of the nostrils there was a diminution of the secretion, and an increased amount of secretion in 4 per cent.; while the redness (colour) of the nasal mucosa was diminished in 18 per cent. of the total nostrils examined.

The reactions on passing from the normal to the hot, dry, hot, medium, and hot, moist respectively, from the hot, medium to the cold, and from the cold to the hot, medium conditions, are also shown schematically on the same chart.

The percentages method employed in making Chart 1 does not bring out the differences between cases of slight or moderate swelling of the inferior turbinates and cases of marked swelling where the increase in the size of the inferior turbinate produced contact with the septum. Chart 2 was made to show the ratio of the cases of marked swelling of the inferior turbinates to the total number of swellings observed. By referring to Chart 2 we see that the number

NEW YORK STATE COMMISSION ON VENTILATION.

Effect of Temperature on Air-passages of the Nose.



Reactions expressed in percentage of total nostrils examined.

COLOUR OF MEMBRANES.

SECRETION.

Cold, 50° F.
Normal, 68° F.
Hot, 86° F.

SIZE OF TURBINATES.

CHART I.

of cases of marked swelling steadily increases as the percentage of atmospheric moisture increases.

This observation would appear of special importance when considered with the clinical findings later recorded in individuals accustomed for long periods of time to hot, moist air (laundry workers).

NEW YORK STATE COMMISSION ON VENTILATION.

Effect of Temperature and Humidity on the Intensity of Swellings of the Inferior Turbinates of the Nose.

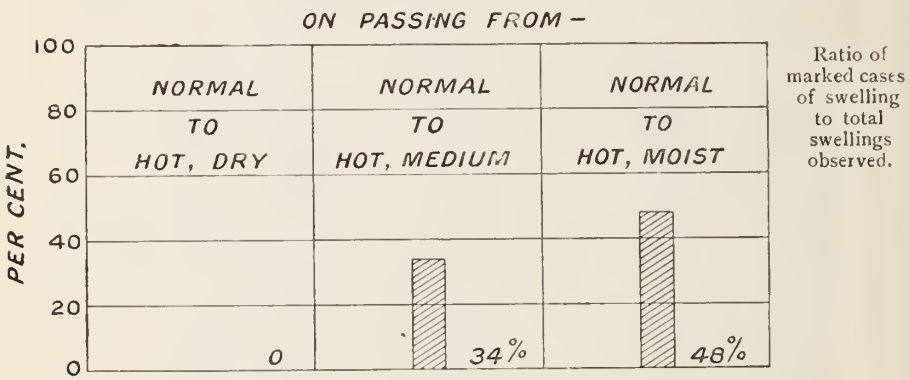


CHART 2.

SERIES B.

Experiments in Air Movement.

The second series of experiments was made to demonstrate the effect upon the nose of a current of air blown directly upon the face by fans. These studies were made in order to obtain information concerning the effects of draughts upon the nasal mucosa. There is such a wide variation of opinion upon the etiological importance of draughts in catarrhal affec-

tions of the upper air-passages that direct evidence upon this subject seems worthy of study.

In Test 1, the subjects were examined in a normal room (68° F., 50 per cent. relative humidity) and then removed to a hot, dry room (86° F., 20 to 30 per cent. relative humidity), where they stayed with electric fans blowing directly on the face as soon as they entered.

In Test 2, the subjects were removed from a normal room to a hot, moist room (86° F., 80 per cent. relative humidity), where they stayed with fans blowing directly on the face.

In Test 3, the observations were made on subjects going from a hot, dry room to a cold room with fans (86° F., 20 per cent. relative humidity, to 50° F., 50 per cent. relative humidity).

Test 4 gives the results of observations on subjects going from a hot moist room to a cold room with fans (86° F., 80 per cent. relative humidity, to 50° F., 50 per cent. relative humidity).

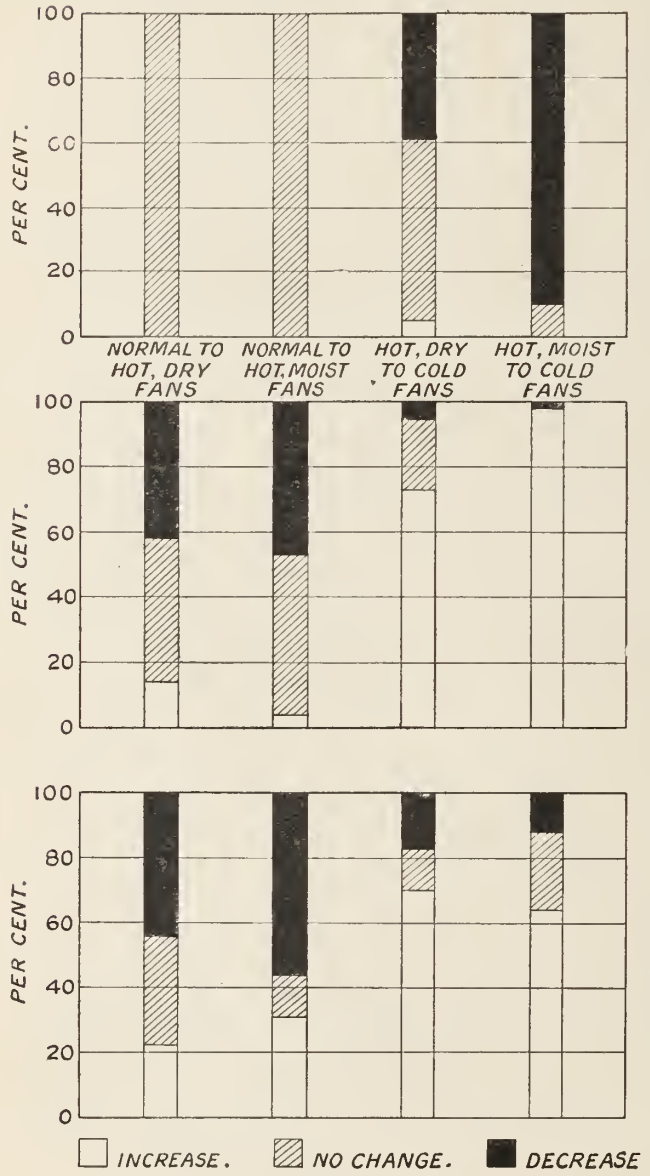
By referring to Chart 3 we see that the characteristic change observed on going from the normal to the hot, dry and hot, moist rooms with fans blowing on the face was a marked reduction in the size of the inferior turbinates accompanied by a diminution in the amount of nasal secretion, slightly more marked in the moist room. This reaction corresponds closely to the results obtained in Series A, where we also obtained a decrease in the inferior turbinates and in the amount of nasal secretion on passing from a normal to a cold room.

This is what we expected to find, as it may be presumed that the physical effects of draughts upon the nasal mucosa is to facilitate the rapidity of heat loss.

In Tests 3 and 4 of Series B, where the subjects were exposed to draughts in a cold room after previous exposure

NEW YORK STATE COMMISSION ON VENTILATION.

Effect of Air Movement on Air-passages of the Nose.



Reactions expressed in percentage of total nostrils examined.

COLOUR OF MEMBRANES.

SECRETION.

SIZE OF TURBINATES (INFERIOR).

CHART 3.

to a high temperature at a low and high humidity respectively, the changes were radically different, showing an almost uniform increase in the size of the turbinates and increased secretion. These results were not anticipated and call for further investigation and corroboration. It would appear from these results that after exposure to high temperatures the effect of draughts is distinctly marked in producing a condition of the mucous membrane especially favourable to the development of infectious micro-organisms.

Series C.

Skin Reflexes.

Examination of Subjects with Body in one room and Head in another having a different Temperature.

A third set of observations (Series C) was made on fifty-seven cases with the body of the subject in one room under certain conditions, while the head protruded into a second room with markedly different atmospheric conditions. The object was to study the reflex action upon the mucous membranes of stimuli derived from the surface of the body.

It was noted that there was a very considerable increase in the size of the inferior turbinates in a large number of cases, quite independent of the atmospheric condition.

A fourth set of experiments (Series D) was attempted in order to elicit further information concerning this reflex action.

In forty-two cases with the body exposed to one temperature, either warm or cold, the feet were immersed in a bath of markedly different temperature. Here, again, the results were inconclusive, the experiments failing to show

any striking or uniform effect upon the shrinkage or swelling of the turbinates and nasal mucosa.

General Conclusions from Series C and D.

The details of the window and skin reflex tests are not given because of the lack of uniformity exhibited in the results.

In general it may be stated that on account of the conflicting results obtained, it is impossible to draw any definite conclusions of the effect upon the nasal mucous membranes produced by changes of temperature exerted upon the surface of the body, as opposed to direct action upon the upper air-passages. This lack of uniformity would tend to show that reflexes from the skin are much less important in producing changes in the nose than is the direct effect of changes of atmospheric conditions acting directly upon the nasal mucosa.

Studies of Workers in Special Occupations.

It was thought desirable to investigate what changes, if any, were produced in the nose and throat by prolonged exposure to extreme atmospheric conditions, and to observe the reactions occurring in such subjects when exposed to atmospheric changes similar to those studied in normal individuals. For this purpose certain occupational groups were selected as follows:—

Series E. Workers in dry heat.

Series F. Outdoor workers.

Series G. Cases of atrophic rhinitis representing steam laundry workers (moist heat).

Series E. Workers in Dry Heat (Boiler-rooms, &c.).

In this group there were four separate experiments.

(1) Twelve observations on six different men on going from a hot, dry to a cold room.

(2) Twelve observations on seven different men on passing from a cold to a hot, dry room.

(3) Eleven observations on six different men on passing from a hot, moist to a cold room.

(4) Ten observations on six different men on going from the cold to the hot, moist chamber.

Series F. Outdoor Workers (Truckmen, Drivers, &c.).

In this group we made four different sets of experiments.

(1) Thirteen observations on seven different men on going from the hot, dry to the cold room.

(2) Fifteen observations on eight different men on going from the cold to the hot, dry.

(3) Seventeen observations on nine different men on passing from the hot, moist to the cold.

(4) Thirteen observations on nine different men on passing from the cold to the hot, moist chamber.

Series G 1. Men with Atrophic Rhinitis as found in Laundry Workers.

(1) Twenty-one observations on cases of atrophic rhinitis on passing from the hot, dry to the cold room.

(2) Eighteen observations on cases of atrophic rhinitis on going from the cold to the hot, dry room.

The results of the experiments on these three occupational groups are shown in the following table. The figures are expressed in percentages of the total number of nostrils examined.

By referring to Table I we find an accurate summary of the results obtained in our experiments. There are several striking differences between the reactions of the normal students (Series A) and those observed in the three occupational groups.

In the first place, we observe an increase in the size of the inferior turbinates of the three occupational groups on going from the hot dry to the cold, the opposite of the reaction obtained in the normal group. The occupational groups, furthermore, show increased secretion under the same conditions. This, again, is contrary to the reaction observed with the normal men.

The two occupational groups Series E and F show practically the same reactions on going from the hot, moist to the cold that they exhibit on passing from the hot, dry to the cold.

In going from the cold to the hot, dry, the characteristic change in the occupational groups is a shrinkage of the inferior turbinates accompanied by a decrease in the amount of secretion, the contrary of the reactions obtained with the normal men.

From these results it would appear that not only are definite changes produced in the nasal mucous membranes by prolonged exposure to extreme atmospheric conditions, but also that as a result of these changes the reactions to variations in the atmospheric environment are distinctly altered from the normal.

Series G 2. The Effect upon the Nose of Long-continued Exposure to Dry Heat and Moist Heat.

After finishing the studies of the direct and immediate effects of various atmospheric conditions upon the nasal

TABLE I.

TABLE SUMMARIZING CLINICAL CHANGES OBSERVED IN THE AIR-PASSAGES OF FOUR GROUPS OF MEN ON GOING FROM ONE ATMOSPHERIC CONDITION TO ANOTHER.

		Number of cases	Change in size of inferior turbinates			Change in secretion			Change in colour		
			Increase	Decrease	Unchanged	Increase	Decrease	Unchanged	Increase	Decrease	Unchanged
Group E.	Hot, dry to cold ...	12	50	25	25	29	4	67	4	4	92
,, F.	,, ,, ,, ...	13	50	27	23	23	4	73	0	23	77
,, G.	,, ,, ,, ...	21	52	37	11	39	21	40	0	83	17
Series A.	,, medium to cold	20	14	43	43	0	33	67	0	28	72
Group E.	Cold to hot, dry ...	12	42	50	8	21	29	50	17	0	83
,, F.	,, ,, ...	15	33	46	21	3	40	67	26	0	74
,, G1.	,, ,, ...	18	48	42	10	3	77	20	65	3	32
Series A.	,, hot, medium	34	46	29	25	9	1	90	24	0	76
Group E.	Hot, moist to cold	11	45	41	14	25	32	43	0	50	50
,, F.	,, ,, ,,	17	67	18	15	44	12	44	0	58	42
Group E.	Cold to hot, moist	10	40	50	10	25	15	60	80	0	20
, F.	,, ,, ,,	13	47	39	14	35	8	57	82	0	18

Group E = Men accustomed to dry heat (furnace men, boiler-makers, &c.)
Group F = Men accustomed to outdoor conditions.
Group G = Men with atrophic condition of the air-passages as found in laundrymen.
Series A = Students used in the tests earlier in the work.

structures we investigated the nasal conditions in workers who, by reason of their occupations, were exposed for long periods of dry heat and moist heat.

The figures elicited are so striking that they appear worthy of serious consideration.

Fifty-eight male students were first examined to select candidates for the subjects used in the experiments already reported in Series A. The ages of these men, the majority of whom were Hebrews, varied from 18 to 23 years. Among the lot were two cases of atrophic rhinitis.

The next group examined consisted of twenty-seven outdoor workers, drivers, teamsters, postmen, &c., ages 19 to 60 years. In this group there were five cases of atrophic rhinitis, one of which occurred in a 60-year-old man, which was probably a case of senile atrophy. Three additional cases had reddened and dry membranes.

Of twenty-six workers in hot, dry air (boiler-room men, engine-room men, stationary engineers, &c.) there were nine cases of atrophic rhinitis, and nine additional cases with reddened and dry nasal membranes, which were not sufficiently advanced to call them atrophic rhinitis.

In our experimental studies we found that the most pronounced immediate reactions were obtained in the hot, moist room. When we came to examine the laundry workers we found that they showed by far the largest percentage of cases of atrophic rhinitis of any of the groups.

We examined forty-six laundry workers while on duty in two steam laundries in New York City and found twenty-one (or 45·6 per cent.) suffering from atrophic rhinitis.

On classifying the employees according to their occupations, it was found that but thirty-three of the forty-six examined worked over the mangles, feeders, &c., where the

humidity was high and where they were actually exposed to escaping steam. The remaining thirteen worked as overseers, packers, &c., receiving and sorting clothes near the doors, where the atmospheric conditions were almost normal. If this group of packers and sorters is excluded from our statistics, we find that of the thirty-three actual laundry workers twenty-one (or 63 per cent.) had atrophic rhinitis.

TABLE II.

TABLE SHOWING INCIDENCE OF CASES OF ATROPHIC RHINITIS.

Group	Number of cases examined	Number of cases of atrophic rhinitis	Percentage of atrophic cases
Students	58	2	3·4
Workers in dry heat	26	9	34·6
Outdoor workers	27	5	18·8
Workers in moist heat (laundries) ...	46	21	45·6

Temperature and humidity records were taken in a number of steam laundries with a sling psychrometer. The average temperature was 75·1° F., and the average relative humidity was 51·3 per cent. On the other hand, the temperature in the washing-room of one of the two laundries where we examined the employees was 69° F., and 83° F. in front of the washing machines of the second laundry. The relative humidity in these two buildings was 71 per cent.

Investigation of the laundry plants shows that steam comes directly up into the faces of the workers much of the time. These steam laundry workers are probably far more exposed to high degrees of humidity than the figures indicate.

We found from a large number of readings that the average atmospheric conditions in hotel and department store boiler- and engine-rooms was 80·2° F., with 26·5 per cent. relative humidity.

Experimental Observations on the Larynx and Trachea of Dogs subjected to Peripheral Stimulation by means of Heat and Cold applied to the Surface of the Body.

We have already alluded to the studies of Rossbach and Aschenbrandt, who found that vaso-motor contraction of the skin vessels due to cold was accompanied by reflex dilatation and congestion of the tracheal vessels, which is thought to be an indirect cause of many of the so-called "cold" diseases.

Meuzer believes that these observations of Rossbach and Aschenbrandt are of little value because the trachea was opened and consequently directly exposed to the outside air.

Through the courtesy of Professor Frederick S. Lee, of the College of Physicians and Surgeons, and with the assistance of Dr. Pike, we attempted to verify these observations.

The experiments were performed on dogs narcotized with chloroform or urethane, and in one instance upon a decerebrated animal.

The room in which the experiments were performed on dogs narcotized was heated to 80° F., so that only warm air could come into contact with the trachea. Instead of opening the trachea, a bronchoscope was used for studying the changes.

Observations were made on four different dogs. The changes observed appear to be too variable and uncertain to warrant our basing any definite conclusions upon them, and do not corroborate the results recorded by Rossbach and Aschenbrandt.

Summary.

Our clinical experiments demonstrate that distinct changes in the mucous membranes of the nose result from alterations in the temperature and humidity of the air as follows:—

(1) Exposure to heat in the majority of normal subjects causes increased swelling, moisture, and redness in the nasal mucous membrane.

(2) Exposure to cold, conversely, causes a diminution in swelling, moisture, and redness.

(3) The effects of exposure to heat are more marked if the humidity of the air is high.

(4) Air currents or draughts directly upon the face materially modify the effects of changes of air temperature upon the nasal mucous membrane. High temperature with draughts result in a diminution of swelling and secretion and low temperature with draughts in an increase in these conditions.

(5) Our clinical and animal experiments fail to demonstrate that any definite reflex effect upon the nasal mucous membrane is produced by exposing the surface of the body to extremes of temperature or to draughts.

(6) Workers subjected for long periods to high temperature and humidity, such as laundry workers, exhibit an atrophic rhinitis in an abnormally large percentage of cases. Workers exposed to drier heat, as in boiler-rooms, also show a high percentage of atrophic rhinitis but less than the group of workers in moist heat.

(7) The nasal mucous membranes of such workers react to changes in atmospheric temperature and humidity quite differently from normal subjects.

Conclusions.

Certain tentative conclusions are suggested as a result of these studies.

(1) The circulatory reactions in the nasal mucous membrane produced by changes in atmospheric environment are too frequent and definite to be disregarded as a probable

predisposing factor in the incidence of bacterial infection of the upper respiratory tract.

(2) Change of temperature is the most important atmospheric condition in the production of these reactions.

(3) The effect produced upon the nasal mucosa by changes of temperature and by draughts is direct rather than a reflex from distant surfaces of the body.

(4) Prolonged and regular exposure to high temperature frequently results in atrophic rhinitis. This condition is a more probable result of moist heat than of dry.

The writers wish to express their appreciation of the assistance of Dr. Robert E. Buckley, who made many of the examinations, and Dr. Bertin Lattin, who searched much of the literature. Our thanks are due also to the following members of the staff of the New York State Ventilation Commission for assistance in keeping the records and carrying on the engineering work: Mr. George T. Palmer, Mr. Buller P. Crittenden, Mr. Robert C. Doremus, and especially to Mr. Joseph Herzstein.

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THE IMPORTANCE OF THE PERSONAL EQUATION IN THE TREATMENT OF THE TUBERCULOUS.

By WILL HOWARD SWAN, M.D.
COLORADO SPRINGS, COLORADO.

In this period of scientific and more nearly exact investigation and treatment of disease, there seems to be a possibility that we may sometimes fail to give sufficient consideration to the personal factors that may have much influence both in the causation and progress of the disease; that the "art" of medical practice may be neglected for the "science" of it. This is more likely to be the case in a chronic affection like tuberculosis; and it is about some of these various conditions that influence the progress of a patient ill with tuberculosis that I wish to speak.

First, I would not be understood as questioning, in the least, the importance of exact knowledge as to the pathology, bacteriology and scientific data that are now or may be available. But I desire to state what is evident to a body like this, that as yet there are no specific agents or means of treating the disease, and that, until such specific means are discovered, our chief aim must be to raise the resistance of the individual to the disease. In other words, we must study and treat the individual who has tuberculosis rather than the disease itself.

Everyone realizes that all cases require an abundance of fresh air, good food, and proper regulation of rest and exercise. But are we not too often content to give our patients this general advice, leaving them to work out details which, for many reasons, they are incompetent to do?

Given a patient ill with tuberculosis, should not our first thought be that he is ill not simply because he has become infected, but because his resisting powers have not been sufficient to overcome the infection? And should not the second step be to hunt out and treat the causes which, in him, have produced such lowered resistance?

Of course, one may have such an overwhelming infection that no resistance can control it.

The question of hereditary predisposition or immunity will not be discussed here; but the fact of the very infrequent incidence of tuberculosis in Colorado and other health resorts where there is an unusually large proportion of tuberculous population is good proof of the reality of acquired personal resistance.

Resistance may be influenced by so many factors in a person's physical, nervous, mental, and moral make-up, by his circumstances as to proper living, and by his adjustment to and view-point of his environment, that much study is often necessary to get at the real situation. But in many cases our success will be about in proportion to the care and accuracy with which we do it.

A person ill with tuberculosis may have reason for lowered resistance in some previous or co-existent disease, and the recognition and correction of these conditions may be the means of giving him the "lift" needed to enable him to overcome his tuberculosis.

Occasionally we are deceived as to the amount of food taken by a patient and it may appear, on careful observation,

that this is the keynote to his failure to improve and perhaps the reason of lowered resistance in the first place.

A man had been sick several years; never very ill, but with very limited strength and always very thin. He was unusually intelligent and there seemed no reason to think that he was not eating abundantly. Careful observation of his diet led to suspicion. He was more liberally fed, with the result of prompt gain of nearly 20 lb. and marked improvement in all particulars.

Numerous instances could be quoted of marked improvement and even cure of tuberculosis after finding and removing the cause of prolonged digestive disturbances and analogous conditions. Mention will simply be made of a few.

A young man had been moderately ill with tuberculosis for several years without material gain. He had more or less indefinite digestive disturbance during this time. Finally a frank attack of appendicitis led to the removal of his appendix. His digestion from that time was greatly improved, he gained weight and strength, and his disease became inactive to the extent that he was able to complete his interrupted college course and has since lived a fairly productive life.

A woman had been thin and weak for several years, unable to eat or digest comfortably, was obstinately constipated and constantly "bilious." A sharp "Lane's Kink" was found and remedied, with the result that she now is largely relieved of her digestive disturbances.

A young woman failed to improve during several years while living under nearly ideal conditions. She constantly had poor digestion, ate and slept badly; was nervous and introspective. She had a material cavity and her disease was slowly progressing. After long and careful consideration, and against the advice of able consultants, she was allowed to take up horseback riding, tentatively at first. There was prompt relief of her digestive difficulties, followed quickly by general improvement. After about two

years she was married. This seemed to improve some nervous complications and she has, for seven or eight years, lived a normal active life in an eastern coast climate, apparently well.

The recognition, during the past few years, of the importance of obscure focal infections in the causation of varied systemic and general diseases has brought help to some victims of tuberculosis.

Two cases, a man and a woman, had failed to improve materially under intelligent and faithful care extending over many months. Both had continuous fever, poor digestion, were sallow, and had moist pulmonary lesions. Finding and relieving an alveolar abscess in one and marked pyorrhœa in the other changed the whole picture. Both quickly lost their fever, ate and digested well, colour improved, cough and expectoration became much less, with drier chest signs, and a gain of about 20 lb. in two or three months in each case.

Similar improvement appears, in many instances, to have been brought about by the removal of diseased tonsils or the cure of diseased accessory sinuses.

These conditions probably did harm, both because of the prolonged slow absorption which occurred from them, and from the fact that they were the starting-points of repeated infections, either of the respiratory tract or of a more general character.

Such occurrences bring up a very important and pertinent question. If the late removal of these depressing lesions has been of so much benefit, how much better that the conditions should have been discovered early, before extensive and perhaps irreparable damage had come to the patient.

The depressing effects of living under unhygienic conditions as to sunlight and ventilation, physical overwork, insufficient rest and sleep, and various forms of dissipation

are so well recognized that they are not likely to be overlooked. But I am quite sure that we often fail to appreciate how frequently weakened resistance is caused by lowered nervous and mental tone from fatigue, worry, grief, discontent, homesickness, and unhappiness from whatever source.

I have never known a person ill with tuberculosis to do well while continuing to be homesick. This is a difficulty frequently encountered by physicians practising at health resorts. It is a condition not always easy of detection; for the homesick patient is very apt to conceal his state of mind with the idea that it is a matter of lack of will-power and that he will fight it out alone.

In my experience it has many times been necessary to send for a patient's friends, or some member of his family, or to send him home in order to overcome this most distressing and depressing malady.

A young woman only moderately ill and apparently living happily and properly with friends from her home town, made no gain during a period of many months. She insisted that she was happy and her friends did not suspect that she was homesick. She ate and digested poorly and lost weight without apparent reason in her physical condition. Finally she admitted that she had been constantly homesick and at times almost desperate. She was sent home, to very unfavourable conditions, where she promptly gained markedly in weight and in all particulars, though eventually she died.

A mother of two small children, whom she had left at home, during many months failed to do well, slept and ate badly and was very nervous, though apparently always cheerful. For a long time she insisted that she was contented and satisfied with the existing plans. Finally she gave way, said she had been constantly homesick and that she must go home without a day's delay. On the trip her train was wrecked and she was subjected to severe nervous

and physical hardship, which produced a condition bordering on collapse. As soon as she was able to travel again, she returned to Colorado to identically the same conditions as before, except that she brought the children, whom she insisted on personally caring for. From that time on she ate, digested, and slept well without medicine, lost all nervousness, and has since been doing much better.

In contrast with the above class of cases, patients are sometimes worried and nagged by their over-anxious and indiscreet family or friends. It may be a very difficult and delicate matter to handle, but often a patient's improvement will depend on the physician's detection and management of these incompatible people. Someone's facetious remark that a physician must also be a policeman if he would successfully treat the tuberculous, contains much truth.

Worry about finances or anything else may do immense harm. Even though we may not be able to remove the cause of the worry, we may help the patient to be philosophical about it and to minimize its importance. The attainment of a cheerful state of mind may be almost as helpful as removing the trouble itself. I have often been impressed with the importance of cheerfulness, content and hopefulness, as a therapeutic measure, as well as the effects of various depressing mental and nervous factors in the causation of lowered resistance. In looking over the histories of 740 cases of tuberculosis treated during the past fifteen years, 129 show reasons for believing that some form of nervous depression was at least an important causative factor. A large proportion of the earlier histories were carelessly taken and give no evidence at all bearing on the subject. I feel certain that it is much more commonly a cause than these figures indicate.

The great mistake and cruel injustice of sending people ill with tuberculosis far from home without sufficient financial

resources to enable them to live under proper conditions and for a sufficient time without worry and embarrassment has often been noted. But those who practise in health resorts still see so much distress from this cause that it cannot be over-emphasized. Getting well of tuberculosis is a long process, and the cost of living and of care suitable to the patient's needs is usually greater in a health resort than in one's own home. A better climate will rarely overbalance lack of proper living conditions or worry and anxiety in obtaining them. Moreover, distinct harm is likely to come from advising a plan which the patient is financially unable to carry out.

The prolonged physical and mental rest and the limitations necessarily imposed during a "cure" sometimes become so irksome as to be a real detriment to the patient. In such cases finding out and making available suitable diversion and mental occupation may be the means of changing failure into success. The prescription must be suited to the individual, else harm rather than good may be done by it.

All these considerations help to show the need of personally taking the histories of our patients and getting into close confidence with them in order that we may know all the factors in their lives which have to do with their powers of resistance. They should also indicate the danger of giving routine directions based on a physical diagnosis alone and of treating our patients as a class. We need to make a kind of social service diagnosis and to do the kind of work done by social service workers if we would most efficiently help our patients in the personal struggle to get well of tuberculosis.

DISCUSSION.

Dr. J. N. Hall (Denver, Colorado) : I have frequently seen tuberculous patients doing badly because they were not able to digest enough food. It is such a common thing to find chronic appendicitis that I have sometimes thought I have found more than I ought to find, and have laid more stress on that particular feature than ought to be done. Years ago I always supposed those people with appendicitis had probably tuberculosis in the appendix. I have been surprised to find out, in almost every instance, that I was dealing with an ordinary chronic appendicitis, and nothing did the patient any good until he got his digestion straightened out. I feel that it is a fair statement to say that a man with tuberculosis who has chronic appendicitis ought to be treated as if he had the appendicitis without any connection with other things. In other words, it is so immensely important for the man with tuberculosis to have a good digestion that we should even take some risks in his operation in order to give him a fair digestion. We might paraphrase what Dr. Bonney once said about tuberculosis, " The poor man cannot afford to have tuberculosis," and say, " The tuberculous cannot afford to have appendicitis."

Dr. Philip King Brown (San Francisco) : I think our experience at Arequipa Sanatorium, where we deal solely with young women, bears out two of the points Dr. Swan has made. First, about 10 per cent. of the patients who go there have to have a great deal of attention given to the digestion, and 5 per cent., before they have been there a year or before they have gone there at all, have had appendectomy either considered or performed. We take a very conservative view of chronic appendicitis and remove all of the unfavourable and variable conditions we possibly can for at least two months before we resort to operative procedures. Although the use of gas and oxygen has reduced the dangerous character of the operation, so that we have not the fear of it we had in the beginning, the getting of a patient to the point where operation seems the lesser of evils is a long job a great many times.

The question of operation adds materially to the expense of care, and not only because operation is a risk to the patient, but also because of the expense we resort to every other means first. They are given a thorough examination, beginning with their teeth, and we have the teeth and tonsils attended to before we take them at all, and special care is given if they have any history of digestive disturbance that is not the ordinary type of disturbance that patients have who are tuberculous.

In regard to what Dr. Hall has said of tuberculosis of the appendix, only in one of our patients has it been found at operation. The vast majority are exceedingly chronic affairs. Chronic appendicitis in a tubercular subject is of apparently more importance than it would be

in an ordinary individual, because of the likelihood of symptoms of constipation and the pain and nausea, besides the uncertainty as to the nature of the process.

That operations can be done even under favourable circumstances with bad result is indicated by one girl we had. She gained 25 lb. or 30 lb. and then reached a stationary period. In it she had an attack of acute appendicitis. We kept her in bed six weeks after the attack before deciding to operate. The loss of weight, pain and tenderness continued. Nothing was found in the appendix other than the adhesions of an old trouble and some apparently recent adhesions. She continued to go downhill, had frequent movements without evidence of ulcer, general tenderness of the abdomen, and later a pleurisy. Five months later she is still below the conditions that held before operation and is still in bed, although the lung condition remains satisfactory.

So ,while we know that occasionally operative interference must come, it is a thing that we feel we ought to be very conservative about.

Dr. W. Jarvis Barlow (Los Angeles, California) : One thing about Dr. Swan's paper that impressed me is the individual treatment of these patients. We cannot impress it too much upon ourselves that all the patients must be individualized, whether at the sanatorium or at home. It may be easier to do this at home, but even at the sanatorium the patients must be individualized. We have got to change our exercise and rest according to the patients, especially with reference to their digestive processes. I found, with surprise, that when I exercised patients whom I thought to be at rest their digestion improved very much, and with it the weight.

In regard to operations for digestive disturbances, as late as five years ago I was very much afraid to have my cases operated upon when there were symptoms of appendicitis. Recently I have changed my views entirely. I think the tuberculous patient stands operation very well. I have been much impressed with it the last year and a half. With three cases I hesitated for six weeks, and when I finally came to it they went through the operation as well as any normal person and afterwards improved tremendously.

The psychology of the tuberculous has also interested me, and I think most of us feel that the mental condition of the patient is such a very important subject in the consideration of the case that it should be taken into account almost immediately.

Dr. John M. Swan (Rochester, N.Y.) : It seems to me the important point in Dr. Swan's paper is the stress he lays upon the mental attitude of the patient. It is not only tuberculous patients that ought to be mentally contented, but patients suffering from any chronic disease. I know that physicians in the past have been negligent of this part of therapeutics. I think this is one of the reasons why the various psychological cults, such as Christian Science and the Emanuel Move-

ment, have had so much success. I think perhaps the reason is that when a physician has studied a case thoroughly and reached a diagnosis he has used up a great deal of his own mental energy, so that it is easiest for him to say to the patient, "You have this or that complaint, and must do so-and-so." He, having reached his conclusion, can outline a treatment, but the patient cannot follow his reasoning, and cannot appreciate that he has, perhaps, used up so much mental energy that he has no more to spend in working out details. This is a phase of our work that we must always keep in mind: the patient, in order to accommodate himself to a disease which he fears, requires pyschological treatment in addition to therapeutic measures.

Dr. G. Walter Holden (Denver, Colorado): Personally, I have found that the patient's mental attitude has a very important bearing on the getting-well process, and is frequently influenced unfavourably by the people at home—the parents, the home physician, and others. As a rule, the parents do not hesitate to take all the family troubles to the patient, and their correspondence is made up largely of the trials and tribulations at home, the financial stress, or the illness of the baby (usually neglecting to state the character or extent of such illnesses), and a hundred and one other little details that keep the patient worried and upset. The patient can be relieved of this in a large measure if you succeed in obtaining his confidence, and can communicate with the home people, telling them how necessary it is that all worry and anxiety be kept from the patient as much as possible. When this is possible the progress is usually satisfactory.

As to the question of diet, that again is a matter of individual study. One person cannot handle starches, and with another you find you have to cut down the proteid diet, so that it is only by individualization that you are able to select a *menu* that will meet the situation. In a small institution it is comparatively easy to individualize, but where you have a large number of patients it is extremely difficult to carry out all the minute details in diet, unless you have the assistance of a competent dietitian.

LUNG INVOLVEMENT SECONDARY TO SUPPURATIVE ABDOMINAL DISEASE.[1]

By J. N. HALL, M.D.
DENVER.

The frequency of subphrenic abscess is probably not appreciated by the majority of practitioners. Certainly the less common instances in which a suppurative process burrows through the diaphragm, and gives rise either to empyema or to perforation into a bronchus or the pericardium, are frequently overlooked. It is these cases that we are to consider to-day.

Since the subphrenic abscess is the preliminary to the suppurative process above the diaphragm we must consider it briefly. An ascending infection from a suppurating retrocæcal appendicitis is a common cause. I have reported[2] nine instances in a series of 500 cases of appendicitis, and do not, from my subsequent more extensive experience, think that this overstates the frequency of the complication.

Christian and Lehr found that subphrenic abscess existed in over 8 per cent. of fatal cases of appendicitis. In the vast majority of cases the right side is involved.

A study of cases occurring through the whole United

[1] Read before the American Climatological and Clinical Association, June, 1915.

[2] *Surgery, Gynæcology, and Obstetrics*, October, 1911.

States would doubtless show that the amœbic hepato-pulmonary abscess is the one most frequently met with. Diffuse subphrenic suppuration is much less often found in this type than in any of the others. This is because it is the rule for the diaphragm to adhere to the liver in such a way that the abscess breaks into the pleural cavity, or more frequently through adhesive inflammation of the pleural layers, into the lung itself. The characteristic anchovy sauce expectoration and the presence of the amœbæ are distinctive. The predominance of right-sided involvement is as marked as in the case of the appendiceal complication.

Gastric and duodenal ulcers frequently cause subphrenic abscess, but this rarely perforates the diaphragm. This is the exact antithesis of the condition in amœbic dysentery, in which we have frequent involvement of the pleura and lung, with infrequent subphrenic abscess. Left-sided subphrenic abscess generally originates from ulcer, although some of the organs to be mentioned later may be the starting-point. The frequent presence of gas is a feature of the abscess originating in peptic ulcer, the gas coming directly from the hollow organs involved, while in the appendiceal cases it is oftener the result of bacterial action.

In a case reported to me by Dr. Thomas H. Hawkins, an abscess resulting from a suppurative salpingitis was finally evacuated through the lung. The spleen, the kidney, the colon, the portal vein, or other abdominal organs may be the site of the original lesion. Traumatic cases have been reported. In acute pancreatitis and in suppurative cholecystitis I have seen the left pleural cavity invaded by an empyema, presumably through the extension of the process into the lesser peritoneal cavity, with inflammation of the diaphragm, and extension to the diaphragmatic layer of the pleura. It is in these cases that the dry pleuritic

friction just above the site of the spleen, of which I have written in the article quoted, is to be found. I regard it as of great significance when detected.

The mere presence of a subphrenic abscess must not be taken to establish its abdominal origin. I have known at least one instance in which at *post-mortem* examination the cause of a left-sided abscess was found to be a perforation of the diaphragm by a neglected empyema secondary to a left-sided pneumonia. I have repeatedly seen pus both above and below the diaphragm in cases in which the subphrenic abscess had perforated and established an empyema above. The perforation of the diaphragm by the needle used to explore an empyema is an occasional cause of a concomitant subphrenic abscess. Archibald's figures would indicate that about 4 per cent. of cases of subphrenic abscess originate from intrathoracic infection. The lymphatic distribution is such that the invasion from the abdomen is vastly more frequent. The right pleura and lung are, therefore, much more frequently involved than the left, since appendicitis and suppurative involvement of the right lobe of the liver cause most of the instances of subphrenic suppuration which bring about the involvement of the respiratory tract. Yet it should be noted that the spread of the process beneath the diaphragm may cause involvement of the pleura on the side opposite to the original abdominal affection, as in the cases just quoted.

I am not aware that gaseous subphrenic abscess has any especial tendency to invade the regions above the diaphragm.

The most frequently found bacterial infection in subphrenic abscess is that by the colon bacillus. The streptococcus, staphylococcus, pneumococcus, *Bacillus typhosus*, and *B. pyocyaneus* are often present. Amœbæ are found

in the dysenteric cases, and the ray fungus and other organisms are occasionally reported.

Diagnosis.—Since the involvement of the pleura or lung can scarcely be suspected until after the time when a diagnosis of subphrenic abscess is possible, our chief attention must be bestowed upon the preliminary condition. And here again the diagnosis of subphrenic abscess often depends upon the recognition of some possible cause in the abdomen. When I state that in one of my cases two different surgeons operated for the relief of a supposed empyema which never existed, both overlooking a glaring history of acute suppurative appendicitis, followed by subphrenic abscess, and perforation through a bronchus, it will be apparent that a definite search for the original source of the trouble is imperative in these obscure cases.

In empyema, unless it is entirely evident that the affection followed immediately after acute pneumonia or broncho-pneumonia, the history should be taken with especial care to overlook no evidences of prior abdominal disease. Not that there is any trouble in the average case in obtaining the history, but merely that such a possibility is often wholly forgotten.

When we have obtained a history of appendicitis, amœbic dysentery, peptic ulcer, pancreatitis, gall-bladder disease, or other similar affection, the first step in the diagnosis of subphrenic abscess is accomplished. Irregular fever, often in the appendiceal cases regarded for a time as typhoid, irregular chills, sweats, leucocytosis, perhaps cough, dyspnœa, pain, and even in neglected cases bulging, redness and œdema of the chest wall, are to be noted. The heart may be displaced to the opposite side. Tenderness and deep pain and discomfort are common. The diaphragmatic movements are abolished. The pushing downward of the liver,

in case the right side be involved, or of the spleen upon the left side, should be noted. Muscular rigidity below the costal border is probably always present.

The higher line of dulness over the liver in front when the right side is involved, and such a pushing upward of the diaphragm behind as to suggest a pleural effusion, are reliable evidences of the presence of the abscess. The X-ray plate demonstrates the shadow of the effusion, and may prove it to be sub-diaphragmatic in location. The fluoroscope reveals the absence of respiratory excursion of the diaphragm. Because of the compression of the base of the lung by the crowding upward of the diaphragm, and the absence of the diaphragmatic influence upon the affected side, feeble respiratory sounds and a lessened vesicular murmur, with even slight dulness, may be detected.

Pleural friction is occasionally to be heard, less frequently pericardial friction. Splashing is decidedly less frequent in pyopneumothorax subphrenicus than in the usual form above the diaphragm. The usual posterior location of the abscess explains the much greater frequency of the presence of signs in the dorsal region, and especially their thoracic rather than abdominal character. The increased area of dulness in the lateral region of the chest is to be found if the abscess be of much size, with crowding downward of the liver if on the right, or the spleen and stomach if on the left. The displacement of liver dulness by tympany if gas be present is of extreme importance in diagnosis, and is usually associated with movable dulness. Subphrenic abscess rarely breaks externally. In one of my cases the abscess ruptured into the colon with fatal results.

In the absence of a definite subphrenic abscess we may be able to detect a liver abscess, especially in the amœbic cases. Its closer relation to the form of the liver, the

enlargement of this organ, and especially the frequently found upward bulge in the liver border either in front or behind, should be considered. Given such a history and some of the findings described, we are ready to consider the possible invasion of the region above the diaphragm. This probably occurs in 25 to 35 per cent. of the neglected cases. It is more frequent in the extra-peritoneal type than in the intra-peritoneal abscesses.

Let us first consider pleural effusion, which so frequently precedes perforation through the lung. In even 50 per cent. of certain series of cases such effusion was found. It may be irritative in origin and serous in character, but may be purulent without any gross perforation of the diaphragm being demonstrable. This is easily credible when we reflect how frequently we find a serous pleural effusion in close relationship with an encysted empyema. I have, in fact, at the present time in St. Luke's Hospital a case in which three different varieties of serous fluid have been withdrawn from the left side around an encysted empyema—clear odourless serum, bloody odourless serum, and clear but most offensive serum respectively. It should be noted that even a pyopneumothorax may exist above a subphrenic abscess without gross perforation of the diaphragm.

The general symptoms after pleural involvement are much the same as those of subphrenic abscess, excepting for the pleuritic signs and cough, which are superadded in many cases. If in doubt as to subphrenic abscess or pleural effusion, the needle must be used freely, the patient being prepared for operation, and in serious cases an anæsthetic being used. If the signs are chiefly anterior in location the exploratory incision is probably always safer, making use of the needle as a secondary procedure. Abdominal aneurysm must be taken into account. The character of the fluid alone may be

decisive. Food particles, coming through a communication with the digestive tract, establish the subphrenic origin of an empyema, excepting in the unusual cases of perforation of the œsophagus into the pleural cavity. This possibility must not be overlooked. I once correctly diagnosed the presence of milk in the right pleura after surgical divulsion of a traumatic œsophageal stricture, the patient having drunk milk freely after recovering from the anæsthetic, with immediate appearance of the signs of effusion in the right pleural cavity. The general history should prevent error in this direction. The greater danger of the use of the exploring needle upon the left side, and the need of emptying the stomach by means of the tube before aspiration, should be mentioned.

In the use of the needle we should bear in mind the observation of Fürbringer that the diaphragm, if perforated, drags the point of the needle downward in inspiration, and hence imparts a contrary movement to the free end of the instrument. Pfühl has described the freer flow of pus from the subphrenic abscess during inspiration, owing to the increase of the pressure within. We are so likely to be satisfied if we find one kind of fluid in one place in the aspiration of the chest, that it is well to recall that when effusion above the diaphragm exists with subphrenic abscess the latter is too frequently overlooked.

The presence of gas or of the colon bacillus in a purulent effusion withdrawn from above the diaphragm strongly suggests, but does not prove, the abdominal origin of the effusion. If in doubt, after drainage has been established, the giving of carmine or other colouring agent by mouth may discolour the discharge if the communication with the digestive canal be still patent. Dr. Edson and I have in St. Luke's Hospital at present a physician who was recently

drained in the region below the left nipple by Dr. W. B. Craig for a colon bacillus abscess which followed a most violent left diaphragmatic pleurisy. In this case the negative result of repeated administration of carmine was most assuring. The usual signs of a pneumothorax are superadded in case gas obtains access to the abscess or the empyema, as is frequently the case. Movable dulness and splashing are especially to be sought.

Acland states that in case of doubt, clubbing of the nails points quite definitely to the supra-diaphragmatic location of the disease. The lung may be involved by the extension of the suppurative process through the visceral layer of pleura. An abscess of the peripheral part of the lung results, preceded or accompanied by a localized pneumonic process. Pleuritic pain and cough are to be expected. They are notable features in the amœbic hepato-pulmonary abscess. Extensive destruction of lung tissue is not infrequent, and expectoration of the products of tissue disintegration may be noted. I have several times found a large superficial cavity immediately above the liver in front, resulting from the great extent of the process. In case of perforation into the lung death may occur from the pneumonic process, from abscess, or from gangrene before the abscess empties itself through a bronchus. In fact, the latter process is probably a function of the milder infections only.

The most striking phenomenon in the whole general process which we are considering is the sudden breaking through of the abscess into a bronchus, with expectoration of the foul pus. This may be in such quantities as to even suffocate the patient. The peculiarly offensive colon bacillus type of odour of the breath is very suggestive. In a case of Dr. George A. Boyd's, the large carrion flies persistently

buzzed about the mouth of the patient when he walked out of doors in summer time, evidently attracted by the foul odour.

In the case of rupture of a septic liver abscess into the pleura and thence into the lung bile may be expectorated with the pus, as are the amœbæ in dysenteric abscess. Pulmonary embolism is spoken of as a complication of subphrenic abscess, coming from the thrombotic process in the veins within the abdomen. A much more striking variety of embolism may occur in the cases of perforation of the lung by the subphrenic abscess. One of my cases, described elsewhere, had septic embolism of the popliteal artery, survived amputation, and suffered two other embolic attacks. The source was evidently the thrombosis in the branches of the pulmonic vein around the large cavity, demonstrated in the lower right lobe. In the *Medical Record* of October 17, 1908, I described "the bowing of the patient with concavity to the right from the cicatricial contraction about the subphrenic abscess and the fistula in the lung." In case the abscess be under the dome of the diaphragm, it may be so far from the surface that it can scarcely be reached by the aspirating needle. In the case quoted above as having been operated twice for a supposed empyema, with entirely negative findings, Dr. H. R. McGraw explored the chest after repeated unsuccessful aspirations, and found the location of the fistula from the old appendiceal abscess into the right lung to be so near the mediastinum that it had escaped all previous attempts at localization.

In another case seen with Dr. W. C. Berlin, a subphrenic abscess burrowed upward along the mediastinum, and a local abscess finally resulted above the right clavicle. The track of the abscess was demonstrable Röntgenologically.

Diaphragmatic hernia with the presence of the stomach above the diaphragm is extremely confusing in diagnosis, but may ordinarily be detected by the X-ray examination, as in the case of a physician recently under my care.

We should mention the rare instances in which the abscess ruptures into the pericardium, as in a case I have reported. Pericardial friction and even splashing may be noted. The complication is one of extreme gravity.

Prognosis.—Barnard states that the mortality in subphrenic abscess is about 16 per cent. under favourable circumstances, and vastly greater in those not receiving surgical treatment. In case of lung involvement, owing to the virulence of the infection and the extent of the destruction of the lung tissue adjacent to the point of perforation, complete recovery must be most exceptional. Yet I cannot agree with Rolleston's statement that death commonly results in two or three months, since I have had three patients who lived from one to ten years each, and two of them were alive when lost track of. All three followed ascending infection from a neglected suppurative appendicitis, and all were proved at operation.

It is becoming widely recognized that the prognosis is so grave that early diagnosis must be followed by prompt operation, even at considerable risk. Empyema must be thoroughly drained in order to avoid the greater danger of later lung involvement. After this occurs surgery rarely offers us any assistance.

DISCUSSION.

Colonel GEORGE E. BUSHNELL, U.S.A. (Fort Bayard, New Mexico): Not being interested in surgery, I cannot comment upon Dr. Hall's valuable paper as it deserves. I was interested in what he said about the effect of subdiaphragmatic conditions in irritating the pleural

cavity. I recently had a case of rather unusual character, where a man was seized with sudden pain in the side. A diagnosis was made of pleurisy, probably tuberculous. He was examined in consultation by a specialist, who confirmed that diagnosis. He came to Fort Bayard. The sounds in the lower part of the lung were very hard to determine, resembling râles. The patient developed a temperature of 103° F. with night sweats, an enormous distension of the liver, and then got well. I think it was a case simply of hepatic congestion without any pulmonary involvement. I would like to ask Dr. Hall if he has had such cases in which irritation of the pleura from subdiaphragmatic inflammation was manifested by sounds resembling râles or friction rubs without the development of a pleuritis?

Dr. PHILIP KING BROWN: I was sorry not to hear Dr. Hall's paper. I have had two recent experiences, both abscesses, one operated yesterday morning, the other a week ago. One was a perinephritic abscess in a woman who had a renal calculus in the other kidney twenty years before. In the kidney on the other side there developed a streptococcic septicæmia and very definite pleurisy above the site of the abscess.

The other was a tubercular abscess of the left lobe of the liver, in which the liver was freely movable for some time, but later there was a leak from the abscess and the liver itself became adherent to the wall and ceased to move up and down with respiration. Very shortly after that the patient developed pleurisy.

Bevan, of Chicago, pointed out at the American Surgical Association, some years ago, that in acute appendicitis pleurisy of the right base behind was not an uncommon symptom. There is no disposition of lymphatics that suggests such an extension, but it is interesting to note that this condition has been noted a number of times.

Dr. JOHN A. LICHTY (Pittsburgh, Pa.): I would like to hear this paper discussed fully, as it is full of interesting information. My experience in this line has not been as great as Dr. Hall's, therefore I am hardly able to discuss it. The frequency with which the Doctor has found subphrenic abscess in his cases of appendicitis is greater than in my experience. I am wondering whether it is possible that he may have a different class of patients to deal with from what I have. In reviewing some 700 cases of appendicitis I do not think I have found empyema or subphrenic abscess any more than five or six times, and in only one of those cases did I find an empyema that ruptured into the lung. I would like to ask Dr. Hall whether he thinks the likelihood of rupture into the lung is greater in these cases of empyema from subphrenic abscess than in ordinary cases of empyema. The subject of subphrenic abscess following lesions of organs in the abdominal cavity is of great interest, and I believe we frequently overlook these conditions. In an article in the *Journal of Surgery, Gynæcology and Obstetrics* a year or so ago, our attention was called to the fact that

subphrenic inflammation can occur without going to abscess and can be very easily overlooked.

Dr. J. N. Hall (Denver, Colorado): In the first place, in regard to the number of cases reported, I think you have to make allowance, when anyone reports cases from any part of the country, for the character of the region in which he practises. I do not think by any means that a man in New York City would see the same material as in the most isolated large city in the United States. You will see that within 600 or 700 miles there is no other city as large as Denver, which is in the centre of an immense country sparsely settled. There are a good many towns that have only a single practitioner who has no one to consult within 50 or 100 miles. When I settled in Colorado I was a railroad surgeon in a small town without a doctor for 100 miles to the west and 139 miles to the east. The territory I covered was as large as the State of Massachusetts. You may be sure that if a case of subphrenic abscess developed so far away it would be well developed before anyone saw it. In New York City that would not occur, because you can get consultants and you would make the diagnosis early. The reason we see so many severe cases there is because of the character of the settlement of the country, and I think, particularly, the great distances between towns, and the fact that a town of 1,000 or 2,000 people will not support the type of medical man who would be apt to spot it early in its course. I might mention, for instance, that one time, years ago, out of three cases in succession, the first and third were appendiceal abscesses operated late, and in each case had resulted in suppuration below the diaphragm and rupture into the right lung. One came from a town in Colorado a long distance away, and the other from somewhere near New Mexico. He had gone on with appendicitis for weeks before the abscess was finally operated. Both of those were of the neglected type, and I should not have seen either if it had been in a large city. This is one of the reasons why we must always insist on having appendicitis operated early. Ninety-nine and a half per cent. of the danger consists in neglecting it until something happens. After one has had these things abundantly, he certainly gets to have a very wholesome respect for what the appendix can do when it is not taken care of.

The question was asked whether empyemas show as great tendency to perforate through the lungs as those which originate subsequent to subphrenic abscess. I think not—not by any means. The subphrenic abscess is a much more virulent abscess. The ordinary empyema is apt to be a pneumococcic affair of mild degree, and the diagnosis more plain. It would not perforate so quickly, and is apt to be recognized more promptly.

As to hearing the sounds above the liver as if pleurisy existed, I have seen so many different types of pleurisy above the diaphragm

that I would not deny that there might be almost any sounds from it. I quoted two cases, one appendiceal abscess, and the other a ruptured gall-bladder in which the left pleura was invaded with a colon bacillus of empyema. The more cases I see, the more keen I am to see the next one.

In regard to the joking remark that Dr. Sewall made when I spoke of some man having a supernumerary nipple, I happened to remark to a doctor that I had come to the conclusion that one man in twenty had a supernumerary nipple. He said he had been practising medicine for twenty-five years and had never seen one. I went to an assistant in his office and asked him if he would mind showing his chest to the doctor, and he showed him two as pronounced ones as I have ever seen. The doctor said, " If you can find two in my own assistant, I will admit that one man in twenty may have one supernumerary nipple."

THE ACUTE INFECTIONS IN DIABETES MELLITUS.

By JOHN A. LICHTY, M.Ph., M.D.
PITTSBURG, PA.

From the very beginning of our knowledge of diabetes mellitus as a clinical entity there has been also a recognition of the peculiar effect of injuries and surgical procedure upon patients having this disease. Many theories have been advanced to explain why a patient suffering from diabetes is a poor surgical risk. By some it has been thought that the difference lies only in the usual administered anæsthetic. It is well known that after general anæsthesia, whether on account of the direct effect of the anæsthetic upon vital organs, among which is principally the liver, or on account of the starvation which usually precedes and follows an operation, acidosis is likely to occur. By these it has been thought if this acidosis could be avoided the operative risk in diabetics would be very little greater than in ordinary cases. Others hold that the difference lies in a disturbance of tissue vitality due to the existing hyperglycæmia. On account of this disturbance bacterial invasion occurs more easily. This view has been largely substantiated by a very interesting and careful work done by Drs. J. C. Da Costa, jun., and E. J. G. Beardsley in the clinical laboratory of the Jefferson Medical College Hospital, and

reported in the *American Journal of the Medical Sciences,* September, 1908, entitled "The Opsonic Index in Diabetes Mellitus." In this work Sir A. Wright's technique, with certain modifications, was adopted to determine the opsonic index to the *Staphylococcus pyogenes aureus,* the *Streptococcus pyogenes,* and the *Bacillus tuberculosis.* Diabetics as a class were found to have subnormal opsonic indices to the streptococcus, the staphylococcus, and the *B. tuberculosis,* the diminution being most marked with the streptococcus and in intensity in the order named. It was concluded that the average diabetic's resistance is reduced approximately one-third below the normal, and in exceptional cases even to two-thirds below normal. Surgeons had practically held this view since the science of bacteriology was known, but it remained for Da Costa and his colleague to show in a rather definite way that it was actually so. The cause of the low opsonic index in diabetes mellitus, as to whether it is due to the hyperglycæmia, to an enzyme not yet recovered, or some other condition not yet recognized, is still a question to be answered. However, through the knowledge of wound infection the mortality of surgical operations in these cases has been very much diminished. At present very few surgeons refuse to do an urgent operation under these circumstances, especially so if they can control the glycosuria to a certain degree. Much has been written upon this subject from a surgical standpoint.

In looking over the literature, little is found, however, which would lead one to think that the ordinary infections which are included under medical diseases are particularly fatal. It is true all text-books refer to the almost invariably fatal issue of erysipelas or of lobar pneumonia complicating diabetes, and also refer to the frequency of pulmonary tuberculosis, but none refer to the danger of the more simple

and ordinary infections. Extensive treatises are published approaching the subject of diabetes from every angle, but no reference is made to the simple acute infections. Of course, the possibility of an infective origin of diabetes is frequently mentioned. Hirschfeld (*Berlin. klin. Woch.*, March 16, 1908) has taken this matter up again by referring to the literature upon the subject and to his own experience. He concludes that cases of glycosuria, which show all the signs of transitory pancreatitis, occasionally develop in the course of infectious processes, especially in persons who are hereditarily disposed to diabetes. The object of this paper is to call attention not so much to the likelihood of the acute infections being an etiological factor in diabetes as to call attention to the fact that they are not infrequently a determining factor in influencing the mortality of diabetes.

My attention was called to this particularly in several cases occurring in my own practice, which I shall now report :—

Mrs. M. W. W., aged 49, wife of a farmer, was referred to me by Dr. William A. Garman, of Berlin, Pa., in September, 1908.

The patient had had the symptoms of diabetes mellitus for one year, as was recognized by her family physician. The amount of sugar eliminated in twenty-four hours was 3·6 oz. The symptoms were not marked. On account of an apparently impending coma she was sent to the Columbia Hospital, where with rest and diet the sugar promptly disappeared and the symptoms subsided. For three successive years this patient came to the hospital every six months, to give us the opportunity of testing her carbohydrate tolerance and directing her diet. Her progress was satisfactory. Her weight four years before the onset of her disease was 185 lb.; at her first consultation it was 136 lb. In January, 1910, it was 151 lb., and she was feeling well, following the duties of her household. About this time she consulted me again, mostly for the purpose of having her diet

enlarged. She was again sent to the Columbia Hospital and her tolerance tested. She could bear considerably more carbohydrates without any apparent danger. While in the hospital she had an acute infection of the naso-pharyngeal mucous membrane. This occurred one day; on the next day she had an intense earache, and an acute otitis media was discovered. The temperature was elevated, the pulse rapid, and the patient nervous and restless. The ear drum was punctured and pus found. On the morning of the third day the patient developed coma, and at night she died. In four days the patient passed from apparently good health through an acute infection of the pharynx to death. I regret to say no culture was made from the pus, and no autopsy could be obtained.

Another case is equally as illustrative:—

Mr. George B., single, aged 26, labourer in the oil fields, consulted me first in May, 1912. He told me that he had been suffering from the symptoms of "sugar diabetes" for three months past. His usual weight was 175 lb.; at this time it was 141 lb. The urine showed the following analysis: Amount in twenty-four hours, 224 oz.; specific gravity, 1033; acid; faint trace of albumin; sugar, 12 per cent., or 26·8 oz. per day. Acetone and diacetic acid were present. At the Columbia Hospital the patient was freed of his sugar in twenty-one days, and was gaining weight. He left the hospital on June 28, about six weeks after I first saw him, and had only a trace of sugar and a gain of 4 lb. in weight. During the following two years, or up to his death, he occasionally consulted me at my office, and several times entered the hospital for a short period to have his carbohydrate tolerance tested. His greatest weight during this time was 151 lb., or a gain of 10 lb. over his weight at the first consultation.

On May 2, 1914, after a month's interval, he consulted me again. He told me he had a cold in the head of a week's duration, and in the past few days coughed considerably. He had a temperature of 100·6° F. and a rapid pulse. Dry râles were found all over the chest, and a few moist râles at the right apex posteriorly. The urine was as follows: Amount in twenty-four hours, 128 oz.; specific gravity, 1030; acid;

a trace of albumin; sugar, 5 per cent., or 6·4 oz. per day; acetone present. I could not persuade him to go to the hospital. He went to his home in the country instead, and died in three days. The report from his family physician was that when he returned to his home he gradually became nervous, then comatose, and died. No chest signs were noticed.

I might mention other cases of similar apparently mild infections, and some of the graver infections, such as lobar pneumonia and acute pulmonary tuberculosis, but they all show the same very rapidly fatal results. In several cases of pulmonary tuberculosis most interesting clinical observations were made. It was found when the restricted carbohydrate diet, upon which the patient usually maintained his weight and feeling of well-being, was relaxed, and an attempt was made to force upon the patient a large amount of general diet, as is usually done in the treatment of pulmonary tuberculosis, the amount of sugar eliminated invariably increased, and the chest signs became rapidly alarming. On the other hand, if the urine could be kept sugar-free, or nearly so, and the caloric value of the diet could be maintained at the normal, or slightly above the normal requirements, other things being equal, the patient invariably improved, as was evidenced by a gain in weight, a diminution of the temperature and pulse-rate, and an improvement of the chest signs. This observation was made so often that it has led me to apply the same principle in the treatment of the acute infections in diabetes mellitus. That is, that in these patients no difference, however grave or apparently trivial the infection may be, the consideration of the diabetic element in the case is paramount and of extreme importance. My experience, in so far as the number of cases of diabetes mellitus is concerned, has not been so extensive as that of other men, some

of whom count their patients in thousands, but in correspondence and conference with them I find their experience is about the same as my own. Of the thirty-three fatal cases of my own series of which I have records eight died of some acute infection. This includes cases of pulmonary tuberculosis and lobar pneumonia.

Among those who have been so obliging as to furnish me with the statistics on their fatal cases of diabetes mellitus, Joslin reports 171 deaths, of which 34 were due to acute infections; Janeway reports 52 deaths, of which 15 were due to infections; Friedenwald reports 30 deaths, of which 10 were due to infections; and Sippey 11 deaths, of which 4 were due to infections. It is thus seen that about one-third of all the deaths are either directly due to or hastened by the acute infections. The recognition of this fact should lead us to warn our patients suffering from diabetes mellitus against the dangers of infections, and instruct them in prophylactic measures. It should also convince us of the necessity of instituting extreme care in treating these cases, even though the infection seems the mildest and most innocent.

DISCUSSION.

Dr. F. M. POTTENGER (Los Angeles, California): I have had some experience with diabetics with tuberculosis. I have been surprised to find these diabetics, when put under regular hygienic treatment for tuberculosis, even without change in diet, lose their sugar. I have seen this in a number of cases. One very interesting case was that of a man who had large quantities of sugar when he entered the sanatorium, but become sugar-free. One day, while playing croquet, a ball struck him on the chin and gave him a severe nervous shock. Sugar reappeared at once. During the last two years I had a diabetic with advanced tuberculosis, who remained free from sugar for more than a year although on general diet. A suggested explanation is that by relieving the tuberculous toxæmia the sugar disappears. Whether or not I am right I do not know.

Dr. JOHN A. LICHTY: In reply to Dr. Pottenger's remarks, I will say that, with very few exceptions, the treatment of diabetes is very much like that of pulmonary tuberculosis, being rest, fresh air, and diet of high caloric value. But if you bring this diet up on the side of the carbohydrates the pulmonary lesion seems to increase, whereas if you bring it up on the side of fats and proteids the pulmonary lesion will be likely to heal as rapidly as under other circumstances. The point I wish to bring out in this paper is the necessity of treating the acute infections in these cases with a great deal of consideration and care. For instance, when a patient with diabetes is taken with such an ordinary infection as a "cold," if you put that patient to bed and take the matter seriously you are likely to save his life, whereas if you allow the infection to go through the various stages, affecting the mucous membranes of the nose and throat, lungs, &c., it is very likely that the patient will go into a diabetic coma and die.

REPORT OF A CASE OF COCCIDIOIDAL GRANULOMA (CALIFORNIA DISEASE). REPORT OF FOUR UNUSUAL ANEURYSMS.

By PHILIP KING BROWN, M.D.

SAN FRANCISCO.

Cases of coccidioidal granuloma, known also as California disease, are very rare even in California, and as far as we know, with one exception, occur nowhere else. Dr. Dickson tells me he has reported nine cases in the recent sessions of the Pan-American Congress. We know these cases are being overlooked all the time in our hospitals. They resemble tuberculosis so very much that they must be classed as consumptives. A differential diagnosis can be made only by cultural or microscopical methods.

This patient is not feeling very fit, and I suppose the ordeal in coming down here is trying. He was a cook on a dining car. He entered the Southern Pacific Hospital after two weeks of cough, fever, and chest pain. Examination showed slight fever, friction rub, râles in the base of left lung and in both apices; a hard, non-fluctuating tumour mass on the head and on his right arm; casts in the urine and a slight amount of sugar; white blood count, 7,200; 2 per cent. eosinophiles. Diagnosis was made clinically without further examination, so familiar are we with these pictures.

Pus from the abscess injected into a guinea-pig resulted in development of typical lesions of coccidioidal granuloma. Cultures were made and found positive. The patient has run a fairly characteristic course. His temperature runs from 99° to 102° F. daily. I think he would do better out of doors, but we cannot board these patients out of doors very well, for they do not want to be classed with the tuberculous patients who are put on the roof of the hospital.

The subcutaneous granulomatous masses were rather hard. They have been curetted and dressed with various antiseptics. This patient has no skin lesions, but several new subcutaneous lesions have developed, and there is an increase in the symptoms in his chest. The skin lesions in these cases are very like the crusty lesions of pellagra. Unusual skin lesions on people who work out of doors in the San Joaquin Valley always attract our attention and cultures are made. They finally break down, and look like syphilitic or tuberculous ulcers. They do not break down as rapidly, in my experience, as syphilitic lesions.

I did not want to speak of the prognosis while the patient was present, but no cases have ever recovered except one where the lesions were in the ankle-joint and the leg was amputated, and one with lesions in the elbow-joint, where prompt resection was done. In neither case were there lung signs. These patients were alive four or five years later. Most cases that come in are primarily affected in the lung. This man's serious symptoms now are lung symptoms. He will die the typical death of a tuberculosis case, and without bacteriological examination one might not recognize the case as coccidioidal granuloma.

I want to report four cases of unusual aneurysms, unusual in symptom and in kind. One patient here has two aneurysms, one of which has been wired.

Aneurysm Descending Arch.

S. G. T. Aged 43. Negro waiter. Denies lues, but his blood shows a positive Wassermann reaction.

Complaint: Periodic diffuse pains in left chest, gradually becoming constant and punctuated by lancinating pain through the cardiac region. Dyspnœa on exertion. Pain is worse in mid-dorsal region around to left axilla.

Examination: Distinct mitral systolic murmur, transmitted outward, but heard all over the heart. No increased dulness made out by percussion at base of heart. No thrill, no unusual pulsation or signs. No changes in radial pulses, no throat symptoms, no tracheal tug or diastolic throb. Slight tenderness over fourth, fifth, and sixth dorsal vertebræ on left side. Blood-pressure, 145. X-ray made diagnosis evident.

Aneurysm Descending Part of Arch.

G. W. Aged 50. Bookkeeper.

Complaint: Constant pain in left side in axilla chiefly; worse when he lies on left side. Admits lues. No cough. Lost weight. Great weakness.

Examination: Tender in mid-scapular region high up, no other physical signs in chest, and nothing detected in lungs.

Diagnosis: Intercostal neuralgia.

X-ray shows aneurysm of descending arch.

Aneurysm Multiple (S. P. Hospital).

O. K. Aged 52. Carpenter. Denies lues, but has a positive Wassermann reaction. Entered hospital February 18, 1915, with pain in right shoulder, beginning six months back. Pulsating tumour noted after few months. Great deal of pain in left mid-scapular region.

Examination: Right pupil larger than left. Pulses at radials not synchronous. No tracheal tug, no thrill, heart broad, confusing murmur heard at base. Subclavian aneurysm wired, and X-ray showed in addition an aneurysm of the descending part of the arch not suspected.

Dissecting Aneurysm (P. K. B.'s Private Case).

E. von S. Aged 47. Travelling man; drives great distances over mountain roads.

Complaint: Diffuse pain in joints and muscles. Has old Pott's disease in lower dorsal region. Denies lues. Overeats and is very fat. While watering garden had sudden weakness in both legs, got into house at once and on to bed before he lost power in legs; not more than two minutes elapsed from onset until power was completely gone. Twenty minutes later on examination *all* reflexes below diaphragm were found abolished with complete paralysis of lower extremities, sensory and motor.

A diagnosis of pressure paralysis from his old Pott's disease was made, with the possibility of an abscess ruptured into spinal canal, for sensory symptoms extended above the old lesion. An orthopædic surgeon was hastily summoned, and arrived an hour and a half later to find the paralysis completely gone.

Patient died suddenly two days later, and at autopsy an extensive syphilitic aortitis was discovered with rupture at the descending arch and complete separation of two of the coats for a distance of 10 in. An eroded area around one of the plaques was evidently the cause of the acute dissecting aneurysm of two days before which cut off *all the circulation below it,* and caused a temporary ischæmic paralysis from blocking of the vertebral arteries, which the recumbent position of the patient corrected sufficiently to admit of some return of the circulation. The lumen of the aorta was reduced to the size of a slate pencil.

These four aneurysms I present simply because they show themselves as clinical entities of great importance, presenting *symptoms,* not *signs.* Positive diagnosis in each case, except the subclavian aneurysm, was made by X-ray examination, or *post-mortem,* and not by clinical examination.

DISCUSSION.

Dr. Ernest C. Dickson (Stanford University Medical School, San Francisco). (With demonstration of gross specimens and microscopic slides): This case of coccidioidal granuloma is one of an interesting group of cases which have, for the most part, been confined to the State of California. As Dr. Brown has pointed out, the majority of the cases have become infected in the San Joaquin Valley. During the past twenty-one years about forty cases have been recognized, but the fact that during recent years the number is increasing much more rapidly than formerly would indicate that the apparent rareness has been due to failure in making a diagnosis.

The most striking feature of the disease is its close resemblance to tuberculosis. In the initial symptoms, in the clinical course, and in the pathological picture, the condition is often indistinguishable from tuberculosis. The mode of onset varies in different cases. In some the initial lesions occur in the skin and may readily be mistaken for the more acute types of tuberculosis of the skin. In other cases the lungs are primarily involved and the patients show the typical signs and symptoms of pulmonary tuberculosis. There is loss of weight, cough, blood-stained sputum, afternoon fever, night sweats, progressive weakness and emaciation, and progressive anæmia. The characteristic signs of pulmonary involvement and frequently of hydrothorax are easily demonstrable. In still other cases the lesions are first manifested in the joints, and they so closely resemble those caused by tuberculosis that in at least three of a series of nine cases which I have reported a diagnosis of tuberculosis of the joints had been made.

The disease may remain localized for a considerable length of time, but early involvement of the regional lymph nodes is the rule, and general infection and death is the ultimate outcome in practically all cases. There are but three known cases in which recovery has taken place. Dr. Brown has already referred to one of these, and in my series of two: one in which the disease was apparently arrested after resection of the elbow, and one in which a spontaneously healed lesion of the lung was found at autopsy in a case which had died after operation for carcinoma of the stomach. In addition to Dr. Brown's case I know of two others, which are still living but which show evidence of active processes, but the remainder have all died, and, with one possible exception, after the development of a general infection.

The etiological factor in coccidioidal granuloma is a mould which has not been definitely classified, but which is somewhat similar to that which causes blastomycosis. Ricketts suggested that the group of Oidia should be extended to include these forms, and until a more satisfactory classification has been established I believe that we should accept his arrangement. The growth on artificial culture media consists of a typical mycelium with aerial hyphæ which bear clamydo-

spores. In the tissues no mycelium is seen, but the organism occurs as spherical bodies with double contoured capsules which reproduce by endosporulation. True budding, such as occurs in blastomycosis, has never been demonstrated.

The origin of the infection is unknown. It has been thought by some to be practically identical with blastomycosis, but the clinical and experimental evidence is all against such an assumption. There is no doubt that the two conditions are closely related, but I believe that it is equally certain that they are etiologically distinct.

In conclusion, I should like to join with Dr. Brown in urging that this disease be kept in mind. I believe that if a routine examination for the spherical bodies of the *Oidium coccidioides* were made in all cases of clinical tuberculosis in which the tubercle bacilli cannot be found, the number of recognized cases of coccidioidal granuloma would rapidly increase.

Dr. W. T. Cummins (Southern Pacific Hospital, San Francisco): I have had the opportunity of seeing a number of cases, clinically and pathologically, of coccidioidal disease, and at the Harriman Laboratory we have been studying the condition from the bacteriological and serological standpoints for the past two years.

Dr. Dickson made mention of the acute type of the disease, which is well illustrated in the first case of our series. The patient was treated for laryngitis which was suspected of being tuberculous. He recovered in three or four weeks and was discharged from our hospital. He was readmitted about three months later with a diagnosis of typhoid fever, which seemed to be confirmed by a positive Widal reaction. In the sixth week death occurred and autopsy showed apparently a diffuse miliary tuberculosis. Histological examination revealed, however, coccidioidal lesions, the identity of which could not have been established without the aid of the microscope. Not having seen a previous case of this disease I consulted with Dr. Dickson, who confirmed my diagnosis. We could obtain no history in this case of skin lesions and we believe that the infection took place through the air-passages. This case has been reported by Dr. Carson and myself.

We have had another interesting case such as Dr. Dickson mentioned—a case with extensive bone lesions. The patient was admitted with a discharging sinus in the great toe. Death occurred after several months had elapsed. A large abscess beneath the scalp in the occipital region was found and this was continuous through the necrotic bone with a purulent collection over the dura. Considerable necrosis of the sternum and the third, fourth and fifth lumbar vertebræ was found as well as caseous lesions throughout the viscera. The double-walled, endosporulating cell was isolated from the pus. This was one of the most extensive cases of coccidioidal granuloma on record.

The case which Dr. Brown has shown you this morning, from the Southern Pacific Hospital, illustrates another type of the disease, in

which multiple peripheral abscesses develop without a septicæmic picture in the early stages or bone lesions.

I am quite convinced that there are more cases of coccidioidal disease in this part of the country than reports indicate. Almost all of the patients have lived at one time in the San Joaquin Valley. Among death certificates made out as tuberculosis there is doubtless a certain percentage of coccidioidal disease. I would emphasize its resemblance to tuberculosis.

Latterly, we have been much interested in the differentiation of blastomycosis and coccidioidal granuloma, and the confusion existing is well shown in the report of MacNeal and Taylor refuting a clinical diagnosis of blastomycosis. We have a small demonstration of specimens of coccidioidal granuloma, blastomycosis, and sporotrichosis at the Harriman Research Laboratory, to which you all are cordially invited.

A CASE OF PULMONARY AND GLANDULAR ASPERGILLOSIS.

By G. W. HOLDEN, M.D.

DENVER, COLORADO.

A CASE of primary pulmonary aspergillosis, originally diagnosed and unsuccessfully treated as tuberculosis, and later tentatively diagnosed as Hodgkin's disease, came under observation at the Agnes Memorial Sanatorium.

Only a comparatively few cases of human aspergillosis have been reported, and most of these occurred in France among the pigeon-feeders and others who handle grain and flour, and the hair-sorters who use rye flour to remove grease from the hair. Sporadic cases, however, have been reported from England, Scotland, and the humid regions of the United States. The extreme rarity of the disease, and the unusual combination of pulmonary and glandular involvement in the case that came under my observation, have prompted me to report it.

Although it has long been known that plants, animals, and birds are directly infected by mould fungi, it was not until Bennett reported the first case of pneumoconiosis in 1848 that these fungi were seriously considered as dangerously pathogenic for man. While some of the moulds which occur as true parasites in man are responsible for a number of common diseases, as favus, thrush, ringworm, and tinea

versicolor, other varieties are truly dangerous, especially when they form poisonous substances (*i.e.*, ergot poisoning).

The fungi pathogenic for man have been divided by Plaut into three groups, according to their pathogenic effects :—

(1) The moulds in the narrowest sense (Mucor and Aspergillus).

(2) The fungus of thrush.

(3) The fungi infecting the skin.

Still divides the genus *Aspergillus* into six species, as follows :—

(1) *A. fumigatus.*
(2) *A. repens.*
(3) *A flavus.*
(4) *A. nidulans.*
(5) *A. concentricus.*
(6) *A. pictor.*

The form most pathogenic for man is *A. fumigatus.* It is very frequently observed in the lower animals and in birds, where it often produces a pseudo-tuberculosis, and it is supposed that the infection may be carried from birds to man.

Aspergillus fumigatus is found most frequently in the ears, nose, and mouth. In the lungs it was thought to be a secondary infection to already existing tuberculosis, until Dieulafoy and others reported cases of primary infection about 1890. A sufficient number of authentic cases of aspergillosis have been observed to make it worthy of consideration in diagnosing obscure cases of pulmonary tuberculosis.

Pulmonary aspergillosis may be either acute—in which case it resembles acute broncho-pneumonia—or chronic, with symptoms very similar to pulmonary tuberculosis. The recognition of the disease depends upon finding the parasite in the sputum. It may sometimes be seen by the naked eye " in the form of little masses the size of a pin's head, resem-

bling the grains met with in streptothrix disease, and, like the latter, consisting of a feltwork of branching mycelium. But more commonly it is first discovered in specimens of the sputum stained with simple dyes," when it appears in the form of branching mycelial threads with spores scattered in and around.

Report of Case.

Case 2435, female, aged 47, married (23 years). Height 5 ft. $4\frac{1}{2}$ in., weight 98 lb. Lived fifteen years in the State of New York, where she was born. Lived in Western Pennsylvania during her married life and up to the time of coming to Colorado for her health in 1913. Four children, aged 21, 19, 17, and 11, and history of two miscarriages, the last in 1904.

Family History.

Father died from abscess of liver. Mother and aunt on father's side died of tuberculosis. One sister living and well. No family history of alcoholism, lues, or cancer.

Previous History.

The patient was never robust. As a child was subject to frequent colds, but never had any disease other than measles, mumps, and whooping-cough. Menstruation began at the age of 11. At the age of 16 had two or three attacks of bronchitis, which recurred every winter thereafter, accompanied by marked chills. Her greatest weight, 137 lb., occurred two years before admission to the sanatorium. Weight at time of admission, 98 lb.

History of Last Illness.

In 1912 developed slight cough, with considerable pain in right arm. There was marked loss of strength and weight. In March, 1913, the right and left cervical glands began to enlarge, followed by enlargement of the axillary glands. A diagnosis of tuberculosis was made, although tubercle bacilli were not found and all other tests were negative; no hæmoptysis. The patient was under treatment for tuberculosis for eighteen months, with no noticeable improvement. On advice of physicians she was sent to Colorado in 1913.

Soon after arrival in Colorado a tentative diagnosis of Hodgkin's disease was made, for which she was under treatment up to the time of admission to the sanatorium. During the winter of 1913 she had œdema of the extremities for six weeks.

At this point I will say that she gave a history of having spent the summer preceding the onset of her disease at a Pennsylvania watering place. The season was an unusually rainy one, and she was constantly annoyed by finding her wearing apparel and shoes becoming mouldy, especially the garments kept in trunks. However, it would be hard to say whether this had any bearing on the case, and I very much doubt its being a causative factor.

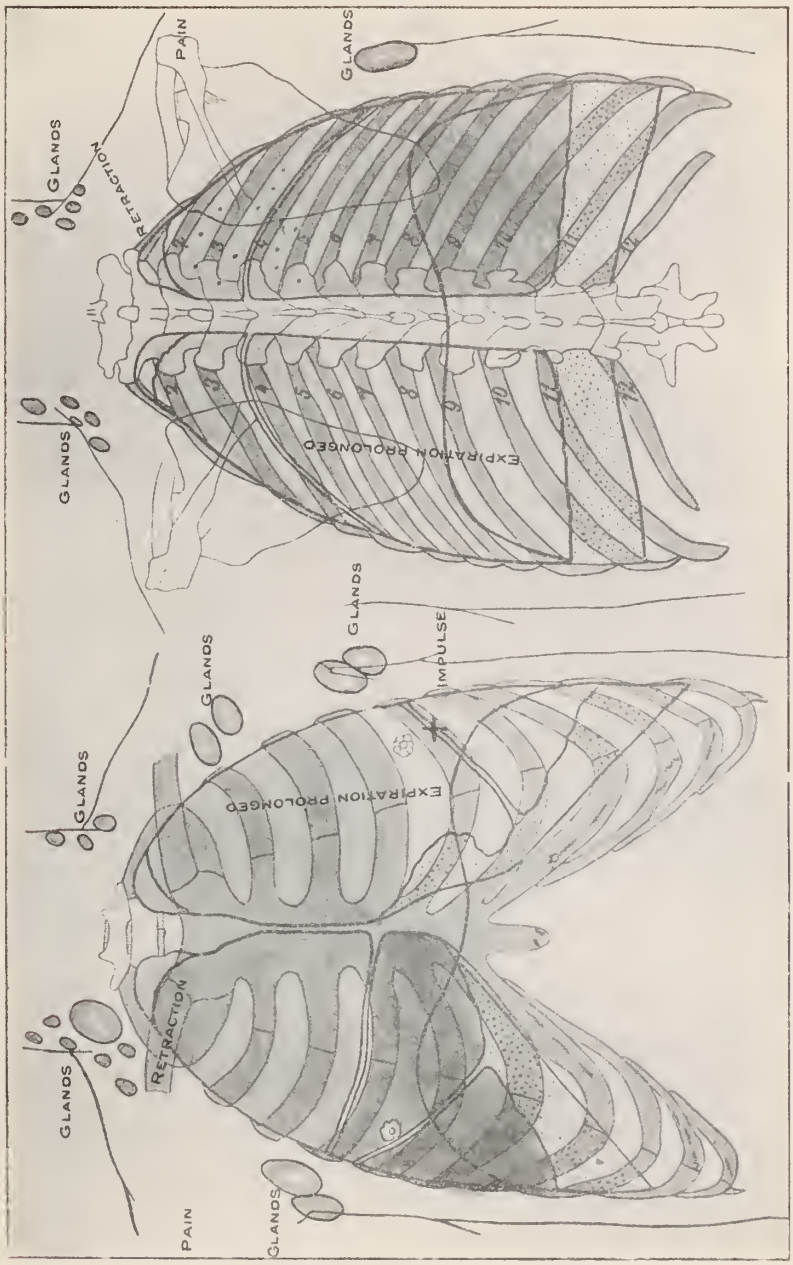
Symptoms at time of Admission.

Admitted to sanatorium August 7, 1914. Temperature, 97·2° to 102° F.; pulse, 90 to 130; respiration, 22 to 28. Weight, 98 lb. The most annoying symptoms were marked dyspnœa, especially when recumbent, profound weakness, a hacking cough, and constant pain in the right shoulder. The cough was of a bronchial character, with a blennothorax, but with scant expectoration (½ oz. in twenty-four hours), and this raised with difficulty. No throat symptoms. Appetite very poor. Digestion impaired. Bowels regular. No night sweats or chills. Both cheeks flushed. Sleep poor, averaging from one to five hours, and not refreshing. Menstruation absent for three months prior to admission.

Examination.

Patient greatly emaciated. Expression drawn and anxious, skin sallow, mucous membranes pale; marked anæmia. Teeth in good condition, tongue red, tonsils normal; nose, throat, and ears negative; voice clear. Oral râles absent, breath sweet, no odour. Fingers clubbed, nails curved and cyanotic. Depression above right and left clavicles marked. Atrophy of right and left scapular muscles marked. Lateral spinal curvature in dorsal region.

Glands.—Right and left cervical glands enlarged, varying in size from a bean to a hickory nut. The enlargements of the right and left axillary glands varied in size from a hickory nut to a small egg (*see* diagram). No thyroid



Location of glands in case of aspergillosis.

change nor enlargement of inguinal glands. The enlarged glands were freely movable under the skin and not tender to the touch.

Chest.—Long, flat chest, with limited motion over the right lung. Chest measurement, 28½ in.; expansion, 2 in.

Right Lung Examination. — Retracted apex, motion limited. Vocal fremitus, and resonance greatly increased, front and back, from apex to base. Dulness over upper portion of lung down to fourth rib, flat from there to base. Bronchial breathing down to fourth rib; breath sounds absent from fourth rib down to base. No râles heard in front. A few moist clicks heard from apex to mid-scapular region behind, after cough only, but not constant.

Left Lung Examination.—Apex slightly retracted. Dulness down to third rib front and angle of scapula behind. Diminished tactile fremitus over this area. Slight bronchial breathing over this area. Breath sounds faint and expiration prolonged—evidently a mild compensatory emphysema.

Heart.—Heart enlarged, and apex well over to the left. Heart sounds of good quality, but faint. At times a faint systolic murmur could be detected.

Abdomen.—Abdomen negative. Liver and spleen normal. Kidneys not palpable. Appendix and colon negative. No œdema of extremities.

Laboratory Examinations.

Sputum.—At first no specimen obtained. Later a specimen showed opaque white masses containing a few epithelia, also a few pus cells. No tubercle bacilli found. The microscopic findings showed a few diplococci and mycelia. The sputum was cultured, and a pure culture of aspergillus found. A second culture revealed the same findings. This was thought to be *A. fumigatus.* In order to confirm our identification of the organism, a culture was mailed to Dr. William H. Park, of the Bureau of Laboratories, Department of Health, New York City, for his opinion. He referred the culture to Drs. Anna Williams and Gurley, who reported as follows:—

"The first specimen examined showed no branching sterigmata, which would classify the organism as *A. fumigatus.* Later specimens examined showed some branching

sterigmata, which would make the classification *A. nidulans.*" In the numerous examinations made at the sanatorium, Dr. Steiner was unable at any time to discover branching sterigmata.

Urine.—Urine slightly cloudy and of a dark yellow colour. Seven hundred cubic centimetres passed in twenty-four hours. Specific gravity, 1025. No odour. Reaction, acid. No albumin, sugar, bile, hæmoglobin, or pus. Urea, 3·1 per cent.; sulphates, ·028; phosphates, ·053; chlorides, 120. Acetone, indol-acetic acid, and diazo reactions negative. Indican, a trace. Microscopic examination of fresh specimen showed epithelia, urates, and many hyaline casts.

Blood.—Blood-pressure, S. 126, D. 84. Hæmoglobin, 59 per cent.

Erythrocytes, per cubic millimetre	5,190,000
Leucocytes ,, ...	13,000
Polymorphonuclears	83·5
Large lymphocytes	8·5
Small ,, ...	4·0
Eosinophiles	1·0
Transitionals	3·0
Mast cells	0·0

Wassermann negative. Von Pirquet skin test negative.

The patient lived fifty-four days after entering the sanatorium. The course of the disease was that of chronic pulmonary tuberculosis, with scant expectoration containing but few pus cells. At no time was there hæmorrhage or blood-stained sputum, as would be expected in aspergillosis.

Digestion was impaired, marked dyspnœa noted, with high afternoon temperature, and the patient slept little. Evidences of pulmonary induration and contraction became more manifest, with increasing marasmus. Death was due to right-sided dilatation of the heart.

Pathological Report.

A *post-mortem* was not permitted, but permission was granted to remove one of the axillary glands. Dr. William

Whitridge Williams examined this gland, and reported pathological findings as follows:—

The gland was of whitish colour and of elastic consistency. Cut surface was of glistening appearance, with a few small, yellowish-white spots. Sections were stained with hæmatoxylin and eosin, eosin and methylene blue, Granis and Loeffler's methylene blue.

There was no evidence of glandular structure. A few small areas near the periphery were filled with small round cells; otherwise the entire parenchyma was replaced by dense fibrous tissue, which in a few small places had become necrotic. Scattered throughout this fibrous tissue were very numerous, rather deeply stained bodies with the following characteristics: At times a short stem ending in a bulbous enlargement, which consisted of a paler centre surrounded by a number of small, dark, round bodies; usually, however, no stem was to be seen, only the bulbs, the majority of which were surrounded by small dots. Between these larger bodies, scattered everywhere in the fibrous tissue, were seen the small dots (spores).

Diagnosis: Aspergillosis, with fibrous transformation of lymph nodes.

This gland substance was also cultured several times for the "corynebacterium" of Hodgkin's disease, always with negative results.

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DISCUSSION.

Dr. HENRY SEWALL (Denver, Colorado): This is a careful report of this case, which I had the good fortune to see before Dr. Holden saw it The X-ray picture taken of this case, perhaps a year before this picture, looked very different indeed. It showed the shadow limited pretty nearly to the upper region. Now it is mainly below and showing rapid distension. Those who, like Norman Bridge, a member of this Society, have devoted much attention to fungus infections of the lungs are impressed with their frequency. The special value of Dr. Holden's report consists in the demonstration of the fungi in the lymph glands and not alone in the sputum, where their occurrence could have been accidental.

THE DIAGNOSIS AND TREATMENT OF CARDIAC SYPHILIS.

By J. M. ANDERS, M.D., LL.D.
PHILADELPHIA, PA.

The possibility of an early recognition of cardiac lues by the history and Wassermann test renders this subject one of special interest, and often leads to remarkable achievements in its treatment. The common occurrence of syphilitic myocarditis, mesaortitis, angina pectoris, and aortic incompetency is now universally recognized. The importance of recognizing the cardiac element at the earliest possible moment is further shown by the fact that involvement of this organ may develop soon after the primary infection without symptoms, which attract attention, until months or even years later. When lues attacks the heart, in young subjects, it may be of hereditary origin. It is also interesting to note that Warthin and Snyder found the *Spirochæta pallida* in the heart muscle, while neither histological lesions nor spirochætæ were found elsewhere.

A statistical investigation into the question of the bearing of syphilis upon the incidence of these conditions, the results of which will be given below, has yielded significant results.

In 1910, Longcope [1] invited the attention of the medical profession to the fact that mesaortitis, a peculiar type of arterio-sclerosis, occasioned by syphilis, was frequently associated with aortic incompetency due to chronic

aortic endocarditis. These conditions may be in turn combined with coronary sclerosis, giving rise to myocarditis and angina pectoris. Among thirty-seven autopsies showing mesaortitis, chronic aortic endocarditis occurred thirteen times, or in 35·1 per cent.

In a series of 930 autopsies at the Pennsylvania Hospital, Longcope found twenty-one cases of chronic aortic endocarditis, unassociated with lesions of any other valves, that occurred in connection with syphilitic mesaortitis. "In these twenty-one cases the lesions varied somewhat in extent in the individual instances, but were always confined, and often sharply localized to, the arch of the aorta" (Longcope). Says Clifford Allbutt [2]: "There will be fewer oversights in particular cases during life if the suspicion that an aortic lesion may be syphilitic is always with us." The inference that cases are of syphilitic origin is often justified by the results of anti-syphilitic treatment, especially in the more acute forms of aortic valvulitis.

Again, of twenty-one cases of aortic insufficiency, eleven, giving a definite history of syphilis or syphilitic lesions (gummata), were discovered at autopsy. Numerous writers have called attention to the frequent association of aortic insufficiency and syphilis, and Citron [3] obtained a positive Wassermann reaction in ten out of sixteen cases of this cardiac lesion, or in 62·6 per cent. Fiessenger [4] obtained a history of syphilis in twenty-eight out of thirty-seven cases of aortic incompetency. Collins and Sachs [5] found a positive Wassermann reaction in ten out of thirteen instances of aortic valvular disease. Babcock [6] records sixteen cases or aortic regurgitation, of which eleven were submitted to a Wassermann test with a positive reaction in all of the cases.

In a recent paper, presented to the College of Physicians, the writer [7] reported the results of his collective investi-

gations into the question of the frequent association of aortic insufficiency and syphilis; they embrace a total of 219 cases, inclusive of the figures cited above. Of these 133, or 60·7 per cent., were clearly due to lues. In a considerable number of the cases of aortic incompetency to be found in the literature no reference to the Wassermann test was noted. Obviously, then, the above figures underestimate the true *rôle* of syphilis in the production of this condition.

Diagnosis of Syphilitic Aortic Incompetency.

The early recognition of this lesion is a matter of the utmost importance to the end that serious damage to the aortic segments may be obviated by intensive anti-syphilitic treatment. On the other hand, if the specific character of the cause be overlooked, or the usual routine treatment of either acute or chronic valvulitis be adopted, these cases may shortly reach a fatal termination.

Syphilitic aortic incompetency may develop subacutely, and an assured diagnosis must rest on the history, supported by a positive Wassermann reaction and other corroborative data, *e.g.*, the age of the patient (usually under the 45th year). In the absence of rheumatism, gout, lead, alcoholism, pyogenic infection, or a predisposing occupation, cases of aortic incompetency should be labelled suspects and given the benefit of vigorous anti-syphilitic treatment. Obviously, in instances belonging to this group, the Wassermann test should be carried out whenever possible before remedial measures are instituted.

There are a few special physical signs which aid in the establishment of the luetic character of the chronic valvulitis. For example, Sachs [8] points out that "enlargement of the left ventricle is generally not marked as in other forms of aortic insufficiency, and, therefore, the capillary pulse and

double sound over the crurales are not so marked." I have observed the presence, to a greater degree, of arrhythmia in cases of aortic incompetency due to syphilis than in those due to other causes.

It is to be recollected that the cardiac lesions of syphilis usually appear within two or three years after primary infection, although, as previously stated, they may develop much earlier.

In cases in which the history in obscure, too much stress cannot be placed on the value of the Wassermann reaction as a means by which to determine the etiologic variety in question.

It must be recollected that aortic incompetency occurring in a leutic subject may be due to causes other than syphilis, more especially if the valvular lesion develops after middle life.

Involvement of the Myocardium.

It must be borne in mind that of all the viscera the heart is among the earliest to become involved in this disease. These early changes consist, as first shown by Adler [9], of peri-arteritis of the coronary vessels. The frequent occurrence of this finding has been confirmed by Harlow Brooks, and later by Warthin [10], who has demonstrated the presence of spirochætes in these characteristic foci, and Wright [11] was able to demonstrate these organisms often in enormous numbers in all of five cases.

Says Richard Cabot [12]: "The permeation of the congenital syphilitic's myocardium with spirochætes (as shown by Warthin and others) makes it probable that in adults as well many cases of myocarditis are due to syphilis." In this connection Babcock [13] pertinently remarks: "Consequently, whenever we have charge of a patient with only the

primary lesion, and still more with secondary manifestation of this insidious and malign infection, we should bear in mind the likelihood of incipient visceral disturbance, and be on the unceasing lookout for signs of functional derangement by which alone, perchance, we may recognize and forestall more serious results."

Diagnosis of Myocardial Syphilis.

When the condition develops in the second stage the onset may be somewhat rapid, or even acute, and also the subsequent course of the complaint.

The principal features are irregularity of the heart's action, tachycardia, intermittence, extrasystoles, and rarely soft murmurs, especially in the mitral area. The symptoms enumerated do not differ from those of myocarditis due to other acute infections, and it has been shown that these indications may all disappear after a few days as the result of energetic anti-syphilitic treatment, a fact that emphasizes the importance of an early recognition of the condition.

Brooks [14] states that, in so far as his observations go, the earliest symptoms are "irregularity of action, more marked when strain is added, as by mild exercises, or through nervous apprehension." The same thing is true of the tachycardia, which is "incited to a degree and by conditions which in the normal would fail to elicit such a response."

Grassman [15] writes: "I have studied 288 cases of secondary syphilis, and in 85 per cent. found disturbances of the rate and rhythm of the pulse, while in 40 per cent. accidental murmurs, usually with dilatation, occurred."

Pain is not a prominent feature in the early phases of the disease, as a rule, although a feeling of thoracic oppression and slight dyspnœa may be noted. In rare instances,

in which extensive lesions are present, actual pain with more or less tendency to radiation may be observed.

In the advanced stages of lues (*e.g.*, tertian and quartenary), the symptoms and signs of cardiac syphilis do not differ materially from those presented by the same conditions, whether the lesions are endo-, epi- or myocardial, due to other causes. Many cases of latent syphilitic myocarditis exist and are unsuspected during life, but are discovered if they prove fatal and come to autopsy. Such cases are often unattended by murmurs and subjective symptoms.

Anginoid pains are not infrequently present, and true angina pectoris may be associated. Due to the myocardial change, dilatation is observed to occur, especially as the result of unwonted exercise, and in some cases, at least, is attended with a murmur. The pains are most probably attributable to the coronary changes, and not, in my opinion, to the ventricular dilatation, since angina pectoris is met with in cases in which no dilatation is found to exist. The observation first made by Runeberg, namely, tonelessness of the first sound at the apex, has since been confirmed by Callender [16] and others. The markedly irregular character of the arryhthmia has been emphasized by Brooks, and is confirmed by my own experience.

The Stokes-Adams syndrome is commonly caused by syphilitic myocarditis or fibroid change in the bundle of His. Robinson found that among sixteen cases of heart-block due to lesions of the His bundle six were of luetic origin. It is to be recollected that some of these cases, at least, will yield to intensive anti-syphilitic treatment.

In concluding the discussion of the diagnosis of cardiac lues, it is to be observed that there is not a single syphilitic lesion of the heart which presents either a characteristic grouping of features, or a single pathognomonic feature,

unless it be a positive Wassermann reaction. Certain clinical peculiarities, however, may serve to arouse suspicion of syphilis. Of these, the tonelessness of the first heart sound, and the decidedly irregular, accentuated character of the arrhythmia should be mentioned. Again, the association of phenomena which point to syphilitic involvement of other organs and structures of the body often throws light on the character of any cardiac lesion that may be present. Lastly, all determining causes of a given cardiac condition other than syphilis should be carefully excluded.

Angina Pectoris.

The writer has collected 270 cases of angina pectoris from the literature, of which only 72, or 26·5 per cent., gave evidence of syphilis. This percentage, however, is much too low, since in 250 of the recorded instances no mention was made of a Wassermann test, sole dependence being placed in the history. The close association of syphilis and angina is amply confirmed by modern authorities. Breitmann [17], of St. Petersburg, writes: "Every case of angina pectoris during the younger period of life is open to the suspicion of syphilitic origin. R. O. Moon [18] has been much impressed with the great predominance of lues in connection with true angina, and has seldom met with a case, since the advent of the Wassermann test, which did not give a positive reaction. Saundby [19] states that congenital syphilis as a cause of angina pectoris is not to be overlooked in young patients.

Treatment.

Prophylaxis.—The fact has long been recognized that a more systematic treatment of syphilis would lessen the incidence of cardio-vascular affections. The spirochætes

display a selective action for the myocardium, and, once embedded here, they are confessedly difficult to eradicate.

Now there is perfect agreement among syphilographers that, when either aortic regurgitation or syphilis of the myocardium of marked extent exists, curative treatment is impossible of attainment, as a rule; hence the obvious necessity for a vigorous and prolonged course of medication during the early stages of this disease, Wassermann tests being made at intervals until a positive reaction is no longer obtained. In this manner, and in this manner only, may we successfully kill the spirochætes before they localize themselves in the cardiac tissues. Physicians should feel themselves charged with a serious responsibility on behalf of the public in regard to this question of the wise direction of the treatment of early syphilis, and realize the greatness of that responsibility, as well as the unanswerable indictment that lies against them if they fail to discharge their plain duty toward this class of sufferers.

Perhaps one of the principal reasons why treatment should be carried out at the earliest possible period after infection lies in the hazard and danger from the administration of salvarsan in cases in which severe forms of cardiovascular syphilis develop subsequently. An investigation into this question has revealed the fact that deaths, either occurring suddenly or after several days, from the use of salvarsan or neo-salvarsan in these cases, are due, in the immense majority of the cases, to myocardial degeneration secondary to coronary lesions.

A sane view of the prophylaxis of cardiac involvement in this disease demands inclusion of the treatment of its secondary manifestations. It is in this stage that their development sometimes takes place, and also that the time of cardiac invasion can be fixed, in some cases at least. Here

it should be pointed out that the cardiac lesions—aortic insufficiency, myocarditis, a certain group of cases of angina pectoris, and aortic aneurysms—are looked upon by many writers as complications of syphilitic aortitis.

In my opinion, when these associated cardiac lesions arise in the acute and secondary stages of syphilis, they are most successfully treated by the alternate use of mercury and salvarsan. The iodides should not be employed in the treatment of these stages, but, as will be pointed out hereafter, are valuable adjuvants quite late in the disease, or after the active syphilitic process has been checked. The most successful method is by the vigorous administration intramuscularly of mercuric salicylate pushed to the limit of tolerance, to be then followed by salvarsan (also given intramuscularly). After the lapse of a few days the mercury must be resumed; and respecting cases that do not manifest the most active symptoms, I am in agreement with Brooks and Carroll [20] in stating that the protiodide administered *per oram* may at times prove entirely satisfactory in its effects.

The writer believes that the method briefly outlined above, especially in acute stages, is capable of yielding more brilliant results than the use of either salvarsan or mercury alone. I am quite aware that many clinicians contend for vigorous treatment with mercury only, believing that the results thus obtained are comparable to those in which both mercury and salvarsan are employed. In these early cases attention to the circulatory disturbances may be additionally demanded. The Wassermann test should invariably be made, and the only criterion of sufficiency of specific treatment is a negative Wassermann reaction. Moreover, the employment of this test from time to time enables one "to determine what success has attended our efforts at eradication of this subtle and obstinate malady" (Babcock).

Furthermore, it should be the rule among clinicians not to place absolute reliance upon a single negative Wassermann reaction, but to repeat the test after the lapse of a couple of months, mercury in some form being exhibited in the meanwhile. At all events, if this procedure cannot be carried out subsequently, then mercury should be exhibited for a year, in order to make thrice certain that a cure has been effected. The failure to obtain a positive result in these cases may have been due either to faulty technique or to the fact that too few spirochætes were present to cause the production of antibodies. I have observed this to be true of cases apparently cured by the use of salvarsan.

On the other hand, early cases of lues are not invariably curable. Again, cases treated with mercury without complete success, owing possibly to the establishment of tolerance toward the remedy, may yield to one or more doses of salvarsan. As illustrative of cases of this kind, the writer has notes of an instance occurring in a male, aged 35, in whom mercury had been employed for a period of four years from the first appearance of secondary manifestations, and it was not until after two doses of salvarsan had been given intramuscularly that a negative Wassermann reaction was repeatedly obtained. My experience coincides with that of Brooks and Carroll, who have found that when treatment by mercury is resumed after a discontinuance for a variable interval of time, a prompt response to it is less likely to occur than in cases in which it was administered without any interruptions.

Treatment of Cardiac Cases in the Tertiary Stage.

The majority of the cases of cardiac syphilis which the writer has treated either in hospital or private practice have been late cases, typical instances for the most part of aortic

regurgitation in decompensation, and equally typical ones of myocardial degeneration independently of aortic incompetency. In the management of these cases, in many of which dropsy has supervened, the usual circulatory indications are to be met as under other circumstances. For example, complete rest of body, hygienic and therapeutic measures, such as digitalis and other cardiants, are all to be enjoined, with modifications to suit individual cases. It cannot, however, be denied that these agencies fail to afford as satisfactory results as in non-syphilitic cases of cardiac disease.

Assuming the Wassermann reaction to be present, how shall the syphilitic element be attacked in these late cases? Shall salvarsan be employed in aortic incompetence after decompensation has appeared? There are two small classes of cases in which the use of salvarsan is, to my mind, quite justifiable. (1) In myocarditis and aortic insufficiency in which the actual condition of the myocardium is indeterminable, but obviously not degenerated to a marked extent, and (2) those cases occurring in patients who urgently demand that something be done to afford them relief from their sufferings. Stone [21] has invited attention to two cases belonging to the latter class in which the distressing symptoms were minimized by the use of salvarsan.

In well-marked cases of syphilitic myocarditis, and in aortic incompetency due to the same cause, it is my custom to give mercury by inunction, and, this failing, by the hypodermic method, a prolonged trial. If this does not yield a favourable result, *i.e.*, a negative Wassermann reaction, then salvarsan is used intramuscularly, at first in small doses. In this group of cases the injections of salvarsan are to be followed by a period of absolute physical rest. Ehrlich, quoted by Held [22], emphasizes this point, stating that it

is an important factor in the treatment with salvarsan of cardio-vascular conditions dependent on syphilis. All such cases should be treated in a hospital, and Held properly recommends that the patient be kept at rest for from twenty-four to thirty-six hours, and in instances of myocardial degeneration not less than two weeks, together with the use of other measures entering into the routine treatment of that condition.

I agree with Daland that the intravenous method of administration of this agent is too hazardous to life to permit of its adoption, and especially is this true in those cases in which decided and rapid invasion of the myocardium has declared itself symptomatically, *e.g.*, in arrhythmia, tachycardia, and the development of fresh murmurs. It should be pointed out that experience has shown neo-salvarsan to be no less apt to induce serious collapse than salvarsan, and the former is not any less painful than the latter, in my experience. Brooks and Carroll are of opinion that "the old salvarsan is more efficacious in the relief of heart complications in syphilis than neo-salvarsan."

Unless a negative Wassermann reaction occurs where salvarsan is administered in the manner stated above, a follow-up course of mercury should be accorded a fair trial, and it may now give a permanent result. To show the degree of improvement gained from any method of treatment, the Wassermann test should be, as already stated, undertaken from time to time.

The iodides are indicated in the treatment of cardiac syphilis after the active process has been checked or has ceased, in order to effect absorption and replacement of tissue, and it is advisable to continue them so long as progressive improvement is manifested. For a period of one year after a negative Wassermann reaction has been obtained

mercury should also be exhibited in these long-standing cases. The dose of the iodides, preferably of potassium, may be pushed to the limit of tolerance, if desired, but I have not seen any better results from this mode of administration than from a maximum dose of 10 or 15 gr. gradually reached after commencing with a 5-gr. dose.

As the result of my limited experience, I have become thoroughly convinced that the majority of late cases of cardiac syphilis do not carry a favourable prognosis as to cure, but some degree of amelioration of the symptoms, and prolongation of life in comparative comfort may be confidently expected in the majority of cases.

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DISCUSSION.

Dr. Henry Sewall (Denver, Colorado) : It is interesting that such an old subject should be made so fresh. There is one point I hope someone will touch upon. On two or three occasions I have had cases in which the apex impulse was very powerful and the first sound practically absent. Those were cases in which I had good reason to suspect constitutional syphilis. I have wondered whether other experiences in that line show any connection between a heart without valvular lesion, apparently organically normal, with impulse at apex, and no first sound detected, and syphilis.

Dr. John M. Swan (Rochester, N.Y.) : I am sorry that I heard only the last of Dr. Anders's paper. In my experience cases of cardiac disease with no definite history of rheumatism or other infection are frequently due to syphilis. In aortic disease particularly, I think one should always be suspicious of syphilis as an etiological factor. In the last two or three years I have had a physician with typical aortic regurgitation under my care. There was no satisfactory antecedent history of the infectious type. I discovered that ten or fifteen years ago he had a suspicious sore on his finger. He had attended a woman in confinement who had a vaginal discharge of a suspicious nature, and I think there is every reason to suspect a syphilitic origin of his aortic disease.

Some cardiac irregularities clear up quickly after the use of iodides. I do not think very much of so-called therapeutic tests, but this is suggestive. I have seen cases in which ventricular extrasystoles disappeared after the administration of sodium iodide.

I think the subject extremely important. Physicians who see a good deal of cardiac disease ought always to be suspicious of syphilis in cases without satisfactory infectious antecedents.

Dr. J. N. Hall (Denver, Colorado) : I would like to say one word as to the frequency of angina pectoris. Benjamin Rush said he never saw but one case and knew only one other reported in his time in America. I think angina pectoris is as well recognized as any disease. It could scarcely get by one who knew anything of the disease. I think that is a fairly accurate statement of the infrequency in this day. It is of much interest to speculate whether the infrequency was due to less syphilis at that time, or whether it is more frequent amongst us because of the rather more intense lives that we lead to-day. Personally, I do not believe there is much doubt that both causes are operative; that is, that syphilis is more frequent than in those days, and the greater strain to which the organism is subjected is in part responsible.

I think there is no question that the generally accepted idea about aneurysm is coming more and more to be that aneurysm in the aorta always means syphilis. I know of whole series where every one had a positive Wassermann reaction.

Dr. F. M. Pottenger (Los Angeles, California): I desire to make one point in the form of a caution. There is a tendency on the part of clinicians in finding a pathological condition present in any organ to conclude that the pathology is that of syphilis if a positive Wassermann is present. This is not true. It has been necessary to fight this same tendency in tuberculosis. A positive Wassermann or a positive tuberculin test only tells us that syphilis and tuberculosis are present somewhere in the body. It does not mean that the pathological process present in any particular organ is of that nature.

Dr. John A. Lichty (Pittsburg, Pa.): I hardly think it would be necessary to call attention to any points in this paper so far as the clinical side of it is concerned, because I am sure there is no one who is more competent to judge the clinical side of this subject than Dr. Anders. There are several points, however, to which I would like to call attention.

Drs. Collins and Sachs read a paper in Washington before the Association of American Physicians, six years ago, at the time the Wassermann was first being used, and it appeared to me to open up a wide field. I saw more cases of syphilitic aortitis after that than ever before. Following Dr. Harlow Brooks's work, I am still more interested. Yet, on the other hand, there are some points that must not be overlooked, and one is that unless we have an autopsy on these cases it is hardly safe to speak absolutely definitely about them, because not all cases where there is an aortic lesion are syphilitic, even though there is a positive Wassermann. That may seem strange, but I have autopsy records to prove this. I collected some of my cases this past winter and reported them before the Biological Society of the University of Pittsburg, demonstrating this very point. There were three most remarkable cases with positive Wassermann. In the first we found a streptococcus infection of the aortic valves. There was little evidence of a syphilitic lesion in the arch of the aorta, but evidence of syphilis in other parts of the body. Yet in the aorta we had only an acute streptococcus infection.

In the second case, with a positive Wassermann I made a positive diagnosis of syphilitic aortitis and found a tubercular pericarditis. The patient was heavy, and it was difficult to examine the heart.

The third man in whom we found a positive Wassermann and an organic heart lesion we found also a streptococcus in the blood-stream. In this case we have not had the autopsy yet, but I am willing to accept the idea that we do not have a syphilitic lesion at the aortic valve.

I think we will have to interpret the Wassermann in the same way we interpret other laboratory tests. Having evidence of aortic lesion and positive Wassermann does not give positive evidence that we have an aortic syphilitic lesion.

I merely mention this, not as a criticism, but simply to point out

that we must not wrongly interpret the value of a Wassermann reaction.

The PRESIDENT called on Dr. McBride to say if he had observed, as he himself had, slight mental failure in early syphilitic infection.

Dr. J. H. MCBRIDE (Pasadena): I cannot say that such is the rule, but it is not infrequent. Slight failure in mental power, or loss of emotional control or eccentricities of conduct or depression are quite common in the early stage of syphilitic infection.

In connection with this subject I recall an investigation of Dr. Campbell, of Edinburgh. He published a good many cases, I think several hundred, of aneurysm of the aorta in which he claimed the lesion was always due to syphilitic infection. I have seen several cases myself of aortic aneurysm in young people where it was apparently due to syphilis.

The report of Dr. Campbell, together with my own limited experience and also the observations of others, have lead me to conclude that aortic aneurysm is generally, if not always, due to syphilis.

Query.—(Dr. SWAN): I would like to ask Dr. Lichty if it is not possible that his patient might have had a streptococcus endocarditis on top of syphilitic disturbance?

Dr. LICHTY: I took this up with Professor Klotz, of the Pathological Department of the School of Medicine of the University of Pittsburg, who conducted the autopsy, and he said it was not a luetic lesion. The difference between a luetic lesion and that due to a streptococcus infection is quite definite.

SOME OF THE ANATOMO-PATHOLOGIC PROBLEMS IN TUBERCULOSIS.

By FRANCIS M. POTTENGER, A.M., M.D., LL.D.
MONROVIA, CALIFORNIA.

Bovine and Human Infection v. Bovine and Human Clinical Tuberculosis.

In this short paper I desire to call attention to several very important problems in the realm of anatomy and pathology which are as yet unsolved, although they have apparently been discussed from nearly every conceivable standpoint.

The question of bovine and human infection has received more consideration during the last few years than almost any other subject connected with tuberculosis, and yet the relationship is far from settled. The scientific world now recognizes it as a fact that bovine tuberculosis infects human beings; but believes equally that the human bacillus is the most common source of infection in human tuberculosis. While the non-pulmonary forms of the disease, those which are particularly prone to infect children, are often considered to be produced by the bovine bacillus, pulmonary tuberculosis, which most commonly affects adults, is considered to be nearly always due to the human bacillus. This has been accepted as meaning that tuberculosis in childhood is conveyed to the human race through milk and meat, while tuberculosis in the adult is probably conveyed from one

infected individual to another; but such reasoning will not hold, for we know that the human being, both in childhood and adult life, is exposed to human and bovine bacilli, and that both gain entrance to the body.

As far as we know, no matter how bacilli are taken into the system, and no matter what kind of bacilli, if in sufficient numbers, they produce an infection. They may be primarily implanted in other tissues of the body, such as seems to be the case from Ghon's reports; but this is only temporary, and, sooner or later, they find their way into the lymph glands and produce lymphatic tuberculosis.

So far as we know, both bovine and human bacilli take the same course. Pathologists who have recovered the bacilli and studied them carefully show that in early life infection is often of the bovine type, while in later life it is almost always of the human type. In this connection I would like to mention that infection in childhood may take place in any part of the body where bacilli are detained in their course, and is as likely, or more likely, to be extra-pulmonary than pulmonary. The reason for this I shall discuss farther on in this paper. It is necessary to cite at this point the very important fact which seems to be well established by pathologists to-day, namely, that the primary infection in the child gives a certain amount of immunity to the disease, and makes further infection much more difficult after a primary infection has once been set up in the body, and the further belief that the later infection of the lung or other portion of the body which manifests itself in adults is in a very large percentage of cases a metastasis from the primary infection which took place during childhood. If, then, both human and bovine bacilli are taken into the body during early life, and if they both produce a specific immunity against themselves, and if they both

become implanted in the tissues and then follow the same course, either going on to active disease, healing, or remaining quiescent for a time, to become active later and spread to other parts of the body; and if the bacillus recovered from the focus in the child is often of the bovine type, while the bacillus recovered from the infection in the adult is almost never of the bovine type, does it not seem that one of two things occurs: either that bovine bacilli find the soil so unsuitable to their growth that unless they produce active disease at once they die out; or that the bovine bacilli, remaining in the human tissues during a period of years, finally adapt themselves to the human tissues, and in the process change their morphological and reactive peculiarities and assume the human type? Such transmutation is far from unlikely. We must look upon the human and bovine bacillus as being different types of the same organism, and only varying because of their different habitat; the one adapting itself to the bovine tissues, and the other to the human tissues. Why could it not be that by subjecting one to the tissues of the other's host over a prolonged period of time that it would take upon itself the characteristics of the bacillus which naturally inhabits that soil?

Primary Focus and Primary Metastasis.

There has been a great deal of discussion on the question of the primary focus in tuberculosis, and also on the primary metastasis from this focus. In tuberculosis of the lungs pathologists have been divided in their opinion as to whether or not the tuberculosis of the peri-bronchial glands is secondary to the pulmonary involvement, or whether the pulmonary involvement is secondary to a primary infection of the glands. This discussion goes on without any particular advantage to the adherents of either theory. Most

observers, however, believe that the glandular involvement is first, and that the pulmonary involvement—that is, the chronic apical involvement which particularly characterizes tuberculosis of the adult—is a metastasis from this.

Ghon has recently, however, revived interest in this important discussion, and brought forth proof, which seems almost incontrovertible, that the primary focus in tuberculosis is often found in the lung, the infection in the peri-bronchial glands being secondary to this. To this discussion I would like to add the following observation, which seems to make it possible to harmonize these differences: There is a marked difference in the behaviour of the infection which takes place in an animal or human being who is healthy at the time of inoculation and one who is already infected. An infection when once present produces a hypersensibility of the cells, and in this way creates a defence against further infection by the same micro-organism. The mucous membranes and other tissues of the non-infected organism show no specific defence, consequently offer little opposition to the passage of bacilli. This we see in the readiness with which bacilli pass through the tonsils of the child and affect the cervical glands, or the bronchi and affect the peri-bronchial glands, or the intestine and affect the mesenteric glands, without being stopped in the intervening tissue. The first important barrier met, prior to the time that infection occurs, is the lymphatic glands, the particular structures which are peculiarly active in the defence of the organism at this period of life. After an infection has occurred, however, and the cells of the body have become sensitized to the bacillus and its products, cellular defence becomes general. Then there is a specific opposition offered to bacilli at every point of entry, the degree varying probably with many circumstances. This was beautifully

demonstrated years ago by Koch's experiments showing that the healthy guinea-pig, when inoculated, shows a slight temporary inflammation at the point of inoculation, which disappears after a short time, but that a very marked involvement of the neighbouring lymph glands takes place, while the inoculation of a guinea-pig which is already tuberculous behaves in a very different manner. This difference depends upon the hypersensitive condition of the body cells. The protective influence of the previous infection shows itself in an attempt on the part of the body cells to limit the infection to the point of inoculation, consequently a nodule appears, followed by inflammation, and after a few days ulceration. The infection being localized is thrown off, the neighbouring lymph glands remaining free from involvement. So, my study leads me to believe, is the case in the natural infection in the human body. If tubercle bacilli pass through the mucous membrane of the tonsils or any portion of the upper air-passages or lungs or lower air-passages, or intestinal tract, they are apt to pass at once to the lymph glands; but, in instances where they fail of this and become implanted locally in the tissues, they form small nodules such as those described by Ghon in his remarkable work. But these foci are extremely small (corresponding to the infection in the healthy guinea-pig at the point of inoculation), many of them not being larger than peas or small beans or marbles. The peri-bronchial glands draining this area, on the other hand, are quite large, and bear the brunt of the infection. Through this infection the hypersensibility of the body cells is produced, and then, when bacilli are carried from these glands to other parts of the body, whether it be the lung, joint, or bone, there is at once a tendency to limit the infection. These metastases usually occur through the blood, and are produced, as a rule, by few bacilli. Escaping into

the blood, the bacilli are diluted and for the most part destroyed. Those which are strained out in the small capillaries to produce a local infection are probably reduced in virulence, and when they come in contact with the action of the sensitized cells their virulence is further limited, so that the lesion which results is either abortive in character or a non-virulent one. This primary metastasis, as a rule, is quiescent for some time before it produces active symptoms, but from it eventually other metastases occur. Even though it may be larger than the primary focus in the lung described by Ghon, it is not followed by marked enlargement of the regional lymph glands. We notice the same freedom from infection of the lymph glands in secondary metastases in the larynx and intestinal tract. Applying this principle of local hypersensitiveness and its effect on attempted inoculation to our study of infection, it offers a key to the determination of the primary focus and the secondary metastasis.

Why the Apex of the Lung is involved in the Adult while any portion may be involved in the Child.

In discussing this question, the very fact that infections in childhood are more apt to be non-pulmonary than pulmonary, while infections in adult life are more commonly pulmonary, and not only pulmonary but begin at the apex of the lung, leads to the natural inquiry as to what difference exists in the conditions which surround infection in child life and those which surround it in adult life. The involvement of the lung, bone, joint, meninges, or other structures than the glands, must be looked upon for the most part as secondary metastases which take place through the blood, although it is possible that bacilli might be taken in through the mucous membrane, particularly those of the intestinal

tract, and be carried through the thoracic duct and poured directly into the blood-stream, to be strained out in any of these parts, without a primary focus occurring in the lymphatic glands. Such bacilli would go through the lung first, and, in case they should escape lodgment in the pulmonary capillaries, might go on and produce infection in some portion of the systemic circulation; but statistics show that bacilli in the child are less prone to find lodgment in the capillaries of the lung than they are in other portions of the body. The fact that the predisposition to pulmonary involvement does not exist in the child, therefore, leads us to the inquiry, is there any anatomic or physiologic difference between the lung in child life and adult life which would make it, particularly the apex, more prone to involvement than any other portion of the body? In answer to this question there are very important changes which take place, and which seem to be fully capable of making this difference in localization. These are the changes which come about in the shape of the thorax and the growth of the lung as the child increases in years.

At birth the jugulum is on the level with the last cervical vertebra; but, after the child has assumed an erect position for a time, the anterior wall of the chest drops. This proceeds rapidly, so that by the time the child is 6 or 8 years of age the anterior portion of the first rib is on a level with the third dorsal vertebra. In this sinking of the anterior portion of the chest the apices of the lungs are crowded together at a time when the lungs as a whole are increasing in size. The respiratory motion is relatively lessened, and this favours retardation of the blood and lymph flow in the apices of the lungs, and in this way favours the implantation of tubercle bacilli at this point. This disposition is particularly exaggerated at puberty, when the lung takes on an

enormous increase in growth, doubling in size within the space of one or two years. The upper portion of the chest being flattened, this increase in growth is reflected in a relatively greater apical compression. As arguments in favour of this being a factor, I would like to call attention to the further fact that the primary metastases rarely occur in the base of the lung where the motion is not retarded; and I would further call attention to the fact that the same localization of carbon takes place in the lungs as of bacilli. While in the child the pulmonary tissue itself is comparatively free, the peri-bronchial glands and subpleural spaces being filled, in the adult the pulmonary tissue itself, particularly in the apices, is studded with carbon, while the pulmonary tissue in the lower lobes is comparatively free.

A Physiological Suggestion for the Cause of the Small Heart.

The small heart in tuberculosis has been the subject of a great deal of discussion from the time tuberculosis first assumed clinical importance. Benecke, Rokatansky, and others observed *post mortem* that the heart was often small in tuberculosis, smaller than in other individuals. Brehmer, being convinced by their observations, decided that tuberculosis was due to the inability of the small heart to properly nourish the lung, and consequently founded a therapy on the principle of improving the functional capacity of the heart. When he established his sanatorium at Goebersdorf he built it at the bottom of a hill, and laid the grounds out in paths representing certain definite grades; and each patient, according to the functional capacity of his heart, was assigned a certain amount of hill-climbing to do. This was guarded most carefully by Brehmer, and a systematic therapy based upon exercise was established.

The fact that the heart in tuberculosis is small has been substantiated later by clinicians, and particularly by the orthodiagraph. The important fact has been brought out that not only is the heart small in late tuberculosis, where it might be due to general malnutrition and degeneration of tissues, but also in early tuberculosis where the tissues have not yet suffered degeneration. In this connection, the small heart is found in persons of normal build, as well as those of enteroptotic build.

Further observation brings out the important fact that there are other conditions in which the small heart is found. It is found in the individual of the enteroptotic build. If we compare the enteroptotic individual and the tuberculous, particularly one suffering from early tuberculosis, we find one condition in common which produces changes in the cardio-vascular system. A striking feature in common is the deficiency in the inspiratory act. In both of these types of individuals we have a deficiency in the action of the diaphragm. In the tuberculous, not only the diaphragm but other muscles of respiration are limited in action. Wenckebach [1] has described this very fully on the part of the patient suffering from enteroptosis. The writer [2, 3] has called attention in numerous papers to the effect of a deficient inspiratory act upon the tuberculous individual.

The maintaining of the normal circulation of the blood depends primarily on the action of the heart itself and the condition of the blood-vessels, but secondarily the circulation is greatly facilitated by the inspiratory act. The inspiratory act opens up the chest cavity, increases the negative pressure in the large veins and heart chambers, and in this way sucks the blood into the right heart. This action at the same time relieves the systemic veins. When there is any deficiency in the inspiratory act, as we find consequent upon a deficient

action of the diaphragm in eteroptosis, and because of the reflex disturbance on the part of the diaphragm, as well as other respiratory muscles in tuberculosis, we have a lessening of the suction action on the circulation and a reduction in the amount of blood delivered to the heart, consequently a reduced amount of blood in the heart, and a reduction in the output of the ventricle. The condition which produces this change being constant in tuberculosis, the heart is obliged to accommodate itself to a lessened content and a lessened output, and as a result of this it becomes smaller in size.

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DISCUSSION.

Dr. MCBRIDE asked if Dr. Pottenger could give a reason for tuberculous meningitis being more common in childhood than in adult life?

Dr. E. O. OTIS (Boston, Mass.): I would be very much interested in the various hypotheses, if you call them that, that Dr. Pottenger has proposed, and he has given us something to think about and investigate thoroughly. I would like to ask one question. How much evidence have we really that bovine tuberculosis is frequent in children?

Colonel GEORGE E. BUSHNELL (Medical Corps, U.S. Army, Fort Bayard, N.M.): With reference to the question of sensitization as the causative factor of the secondary infection, I can hardly agree with Dr. Pottenger, for this reason, that it is not true that the primarily infected animal when reinfected necessarily gets a more extensive infection. The result of the second infection depends upon the degree of immunity that has been acquired from the first infection. If a

primarily infected animal is reinfected in two or three weeks the effect of the reinfection will be well marked. If the reinfection is performed in three months after the primary infection it may or may not produce distinct tuberculous lesions. If a year elapses between the two infections and the primary infection has been small, so as to produce an immunity rather than a generalized tuberculosis, there will be practically no result from the reinfection. In Roemer's experiments sheep primarily infected with human tubercle bacilli were injected with large doses of virulent bovine bacilli a year or more after the primary infection, with the result that some of them died immediately with symptoms of anaphylaxis. Those that survived were as well as ever in a few hours, the second infection producing no tuberculous lesions.

If you assume, as I do and as Dr. Pottenger does, that the great bulk of infections occur in early life, that immunity of a certain type develops and persists, and that tuberculosis when it develops in adult life usually shows considerable immunity—that is, it is a localized process which tends to fibroid changes, does not readily extend, and may be readily cured—if you assume the presence of that type to be evidence of immunity, you have then long passed the stage of extreme sensitization. If the individual had been reinfected a few months after the first infection, you might have the phenomena of sensitization. If the reinfection occurs one, ten, twenty years later, it is quite improbable that they will be present. Cellular immunity is shown by clinical facts to be primarily, or in the first stages of its development, a state of hypersensitiveness, later it is insensitive. This is shown, among other things, by the facts of tuberculous bacteræmia. There seems to be no doubt that tubercle bacilli are to be found at times in the blood of persons who have little or no clinically active tuberculosis. The bacilli circulate in tissues so thoroughly immune that they do not have to react, do not have to develop antibodies, but are simply incapable of being attacked.

I have elsewhere developed a theory as to the cause of the localization of tuberculosis in the apex. My view is that the child gets the early infection, and that the future fate of the individual is dependent upon the size of that infection or upon the number of repeated small infections which may take place before the state of immunity is well developed. If the child has a large reservoir of tubercle bacilli in the bronchial glands he will be more apt to develop pulmonary tuberculosis in his adult life than if he has a smaller one. The large infection interferes with the health of the organism and renders the physical development defective, as manifested by the small heart and the paralytic thorax. The chest shoots up, as Dr. Pottenger has described. It shoots up higher when the patient is not strong and well developed, and you get the long slender chest instead of the short thick chest. The infection once established never altogether ceases. The bacilli collected in the bronchial glands, if there is loss of immunity, will

grow by continuity in various directions. The X-ray shows they more frequently grow downward in the child. The child usually has tuberculosis of the lower lobe if the disease affects any superficial part of the lung, whereas in the adult extension is practically always upward. My notion is, in brief, that the tubercle bacilli work from the bronchial glands along lines of least resistance until they finally reach the apex by continuity, being enabled to establish themselves there by the lack of mobility of that part of the lung in the adult.

Dr. W. JARVIS BARLOW (Los Angeles, California): I am glad to say I find Dr. Pottenger's paper most interesting. I do feel that this is a Society where such papers should be brought out. I do not agree on the theory about the heart, yet I do not know that I have anything new to offer there. I do feel, as Dr. Otis does, that I do not know much about bovine and human bacilli. I would like Dr. Pottenger to explain how he can tell the bovine from human infection in his cases.

Dr. HENRY SEWALL (Denver, Colorado): If I may be permitted to say it, we are using words which we and nobody else understand. Sensitization, for instance. We have an idea when we use it, but anyone who has done immunization experiments realizes that the word is a "big smoke" and we do not know the fire. A year ago, in company with Cuthbert Powell, I did some experiments on guinea-pigs, introducing horse serum into the nose. The phenomena of sensitization, anaphylaxis and immunity were produced in those beasts. We found it quite possible to make a guinea-pig apparently immune without having gone through a stage of sensitization, if sensitization means hypersusceptibility. The point I want to bring out is, that in my own personal observations there seems to be an insensitizing genius as well as there is a sensitizing genius. You throw an antigen into an animal, and he may at once resist and never be disturbed by it. You take another animal, and perhaps its case is illustrated by the history of the fall of man. He has got to suffer in order to get his immunity. Here is a creature who has reached immunity without suffering, without sensitization, without throes, and another reaches the same stage of immunity after being brought to the verge of the grave. That you can demonstrate by experiment. A year ago it was possible to give forty guinea-pigs these serum droppings in the nose and one-half of them would stand a tremendous intravenous injection sixteen days after the last injection of serum. This year the same experiment has been repeated, and every pig would have died under the same conditions. They all were sensitized. It puzzled me to know the difference. Then I found that last year I gave a little less of the serum in each dose than this year.

Dr. Pottenger spoke about massed bacilli. I think very likely therein lies the crux of the situation. If you get millions of tubercle bacilli, you are gone. If you get ten, you can beat it.

Dr. JOHN M. SWAN (Rochester, New York): The small heart is

found in other conditions than tuberculosis. In dysthyroidism we frequently find, on X-ray examination, that the heart is small. Bauer and Helm have published the results of orthodiagraphic studies in cases of dysthyroidism. They found the small heart a common condition, even in cases in which, by clinical methods, an enlargement of the area of cardiac dulness could be determined. They make the claim—and it seems to me that it is quite well founded—that it is a degenerative condition, congenital, and is perhaps one of the reasons why thyroid disturbances, tuberculosis, and other infections occur so readily in such individuals.

Dr. F. M. POTTENGER (Los Angeles, California), closing discussion: I have brought four distinct subjects before you, any one of which would merit a long discussion, consequently it is impossible to do more than touch upon each of them. If I could discuss them more completely it would be more convincing.

Regarding the question of sensitization, as Dr. Sewall says, we are talking about things of which we know little or nothing; at the same time, it is necessary to use these terms in order to define and describe a conception. In this discussion I will displace the word "sensitization" and say "defence," because this represents my idea. In advanced tuberculosis we know that there is a very strong specific defence because an individual will have millions and millions of bacilli passing through his air-passages without infection occurring, and may also swallow millions daily without implantation occurring in the walls of the intestines. This can only be accounted for by supposing that the body cells are endowed with a very high degree of specific defence.

The child is born with general defence but probably little or no specific defence; but, after fighting different inoculations of tubercle bacilli the cells take upon themselves the power to produce a special ferment or special antibody designed to combat the tubercle bacillus, and we say that they are sensitized to the tubercle bacillus. Whatever this process is, my conception is that the cells are changed, so that when tubercle bacilli pass out into the lymph- and blood-stream from some nidus in the glands or other body tissue, or when they are taken in from the outside, if an attempt is made to settle in the tissues, they meet cells which have a specific defensive power, and these cells produce antibodies which tend to destroy the bacilli *in situ*. This is the conception that I had when I used the word "sensitization" in my paper. It is different from the hypersensitiveness mentioned by Colonel Bushnell, which is supposed to mean increased susceptibility. There is a difference of opinion as to whether a condition of hypersensibility means that the organism is more, or less, susceptible. There is a confusion of terms in the discussion and different observers are talking about entirely different things. When we speak of sensitization we are not discussing the question of a child who has not previously been infected, but one that has come in contact with tubercle bacilli a few

or many times and has more or less successfully withstood the infection, so that, during that period he has been able to develop a specific property on the part of the cells whereby they withstand larger and larger doses of tubercle bacilli.

Freund's theory of why the apex is involved is a double one. First, because of the shortening of the first rib; and secondly, because of the ossification of the first costo-sternal joint. Both of these act, according to the author, by interfering with the aeration of the apex. According to my ideas, ossification of the costo-sternal joint is most probably secondary, due to the lessened motion of the side and the reflex trophic disturbances in the articulation. Shortening of the first rib when present acts simply as a part of the general compression due to the flattening of the upper portion of the chest as a result of man's attaining the upright position.

Dr. Otis has asked what evidence there is of bovine infection in childhood. His question implies an uncertainty in differentiating between the bovine and human types of the bacillus. I take as evidence the work which would be accepted by the Imperial Board of Health of Germany, the Health Board of England, and such other observers as W. H. Park and Theobald Smith in our own country. Through careful examination and proving the bacilli culturally and morphologically, and by inoculation experiments, they have arrived at opinions which I think can be fairly accepted. The cases accepted by the Imperial Board of Health of Germany should have special weight, because these workers practically all belong to Koch's school.

In answer to Dr. McBride's question, why meningitis is more frequent in childhood, I cannot answer. My supposition, however, is that prior to the time the child assumes the erect position and the flattening of the thorax compresses the apex in the child, tubercle bacilli are as apt to be implanted in one organ as in another. It may occur in the bones, lungs, joints, glands, meninges, or in any part of the body.

Regarding the animal experiments to which Colonel Bushnell refers, I would like to make the following observations. A great deal of animal experimentation is unnatural. We are placing entirely too much confidence on unnatural conditions which arise in animal experimentation. Clinical men are afraid to record their own observations and assert their ideas, and prefer to take artificial experiments as being more convincing than their own observations. All animal experimentation will bear close scrutiny. Cornet brought forth the main proof upon which the inhalation theory of tuberculosis is based by putting guinea-pigs in a box filled with dust laden with tubercle bacilli and allowing the pigs to breathe it. Flugge proved his droplet infection by confining guinea-pigs in a box and having patients cough over them. Calmette proved the alimentary route of infection by feeding animals with large doses of tubercle bacilli made into an emulsion with butter.

Every one of these experiments is open to criticism, for they are unnatural. They only prove that when tubercle bacilli are brought to the animal in enormous quantities in these ways that he is unable to withstand them, but prove nothing as to the natural mode of infection as it takes place in every-day life. From the clinical standpoint they are worthless. We are dealing with human tuberculosis, which is a very different process from experimental animal tuberculosis. We are dealing with the human organism which begins to come in contact with tubercle bacilli soon after birth and gradually builds up a specific defence against them, so that by the time adult life is reached, the time when the disease begins to spread and form our chronic pulmonary tuberculosis, a high grade of immunity has developed which totally changes the pathological picture. In the guinea-pig, however, we are taking an animal which is highly susceptible, injecting doses unusually large and in an unnatural manner, and then we are trying to solve our problems in clinical tuberculosis by this unnatural artificial tuberculosis developed in the animal. The two are not comparable. It is time for clinical men to look at these questions from an analytical standpoint and not accept all experimental work that is being done as being greater and more convincing than their own clinical observations on man.

Referring to the experiments of Roemer mentioned by Colonel Bushnell, these show that a guinea-pig, which is already tuberculous, on being injected with a second inoculation of tubercle bacilli develops a second disease which is modified by the first infection. The second infection takes upon itself more chronicity, and even in the guinea-pig will at times go on to cavity formation, which means a chronic process. Thus, from the prior infection, even if the pig should eventually die, an immunity has developed which changes the course of the second infection. This same thing is true in human tuberculosis. Metastases which occur in the lungs, bones, and joints in later life are very different processes clinically from the early rapidly destructive processes in the unimmunized child.

I do not wish to be understood as saying that the specific defence of the body cells is a factor in causing localization of tubercle bacilli in the apex. On the contrary, I think it has a great deal to do with preventing infection from occurring, the same as it does in any other part of the body. I also think it has a great deal to do with the form which the secondary metastases take.

Clinically, we have been making a difference between tuberculosis in the lungs, glands, bones, meninges, and joints, considering the pulmonary form the typical and the others atypical. The reason for this is probably because we are comparing two different conditions. My conception is that most tuberculosis spreads in the body through the blood, and that the resulting infections distant from the primary infection are carried there through the blood-stream. Taking place

after infection has occurred, the blood contains greater or lesser quantities of antibodies, and the cells have developed their specific defence, consequently, when the bacilli become implanted there is a tendency to ward off infection at the point of implantation. This shows itself in what we might call an abortive infection or an infection of low grade, usually, at first, fibroid in character. True, the bacilli in this infection may go on to the development and spread of the disease.

Another type of secondary metastasis is that which occurs near the hilus, where the tubercle bacilli probably escape from the glands into the large lymph channels, and, in spite of the large quantities of lymph present, make their way against the lymph-stream into neighbouring tissues. Such an infection is, as a rule, mild in character.

Regarding the small heart being found in other conditions, as mentioned by Dr. Barlow, that is the fact which I take as the key to the situation. The small heart is found in cases of enteroptosis and general asthenia. The factor which is present in common in these different conditions is the deficient inspiratory act. We know that the heart has the power to adjust itself to different conditions. Moritz speaks of its ability to enlarge and decrease physiologically. The important point which favours the explanation which I have given is that it is not only found in advanced cases, as it would be if it was due to malnutrition, but it is found in robust individuals who are suffering from early lesions. True, in some instances the decrease in the size of the heart is so little that it cannot be recognized; in others it is very marked. I have discussed this more fully in other papers, which will soon be published.

THE RELATION OF THE SPLEEN TO TUBERCULOSIS AND FAT SOLVENTS.[1]

By WM. CHARLES WHITE, M.D.
PITTSBURG, PA.

Last year we presented a paper before the Association of American Physicians [1] on some changes produced in tuberculous rabbits by inhalations of benzol (C_6H_6). These changes were anæmia, severe leucopenia, profound bone-marrow changes, and enlargement of the spleen.

The tuberculosis of the rabbit had been produced by the injection of 1 mg. of bovine tubercle bacilli in emulsion into the ear vein. This produced a pulmonary tuberculosis, and very frequently splenic and kidney tuberculosis of a more or less severe degree.

The benzol was given every second day, from a glass cone until complete anæsthesia was produced, as described in the above article [1].

We were inclined at that time to ascribe the blood and marrow changes to the benzol, but were unable to say whether the benzol alone would produce the same result. We had been mainly interested in the changes produced in a tuberculous process in the lungs by the inhalation of a fat solvent, and so had kept no control for the benzol alone. We are now in a position to say, however, that we have not been able to produce in well rabbits any anæmia, leucopenia, or splenomegaly by inhalations of benzol alone.

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[1] From the R. B. Mellon Laboratory of the Tuberculosis League of Pittsburgh.

Table I.

Blood Counts of Rabbits submitted to Benzol Inhalations for Three Months.

	Number of rabbits	Leucocyte count			Red blood count		
		Lowest count	Highest count	Average count	Lowest count	Highest count	Average count
End of first week	4	6,000	13,000	9,550	4,488,000	5,792,000	5,125,000
,, second week	4	8,000	11,000	9,300	5,024,000	5,504,000	5,263,000
,, third week	4	6,400	9,000	7,200	5,120,000	5,624,000	5,368,000
,, fourth week	4	7,000	7,400	7,200	4,576,000	6,144,000	5,622,000
,, fifth week...	4	6,800	8,800	7,700	5,504,000	5,920,000	5,714,000
,, sixth week	4	7,200	8,800	7,800	5,168,000	5,824,000	5,666,000
,, seventh week	4	6,800	8,000	7,850	4,744,000	5,312,000	5,080,000
,, eighth week	4	7,000	8,400	7,800	3,520,000	6,400,000	4,622,000
,, ninth week	4	6,200	6,500	6,800	4,560,000	4,960,000	4,762,000
,, tenth week	4	6,800	8,600	7,860	4,000,000	5,080,000	4,380,000
,, eleventh week	4	5,700	8,800	7,250	3,760,000	4,960,000	4,460,000
,, twelfth week	4	6,700	7,700	7,250	4,320,000	5,200,000	4,706,000
,, thirteenth week	4	7,600	8,500	8,000	4,344,000	4,932,000	4,582,000
,, fourteenth week	3	8,600	9,600	9,000	4,864,000	5,000,000	4,920,000
,, fifteenth week	3	8,800	9,400	9,300	4,200,000	4,848,000	4,516,000

A series of five rabbits received benzol inhalations over a period of three months every second and third day, with no apparent change in their constitution. At the end of that time they ate well, and were apparently normal at death. Their weights before and after treatment appear in Table III.

So striking was the contrast between the normal rabbits receiving benzol and the tuberculous rabbits receiving benzol, that the question arises whether in the cases studied by Selling [2] there may not have been a tuberculous focus present which, by its combination with the benzol fumes in which the patients worked, produced the severe changes in blood and bone-marrow which he found, and which were present also in our tuberculous rabbits.

Enlargement of the rabbit's spleen is not infrequent as a result of tuberculosis. Such enlargement occurs as a result of tuberculosis of the lungs, but is most pronounced when there is also tuberculosis of the spleen.

In the following Table II it will be seen that the spleen varies from a normal size in a case with few tubercles in the lung to almost double that size where the tubercles are abundant.

In the fourth table is given the measurement of the spleens of those animals which received benzol for three months by inhalation. They are normal in size for rabbits of 1,700 to 2,300 grm.

In the third table is given the size of the spleens, a rough estimate of the amount of tuberculosis in spleen, lungs, and kidneys, and the number of inhalations and the kind of fat solvent used.

It will be seen that while the inhalation of a fat solvent has no constant action, yet our largest spleens were found in tuberculous rabbits receiving inhalations of ether or benzol, one spleen reaching the enormous size of 13 × 3 cm.

Where the tuberculosis is small in amount there is, as a rule, no serious enlargement. This may be caused, however, by the short time (twenty minutes in two or three days)

TABLE II.

Tuberculosis only.

	SPLEEN		LUNGS	KIDNEYS
	Size in centimetres	Tubercles	Tubercles	Tubercles
1	5·0 by 0·8	None	Few	None
2	6·0 ,, 0·2	Few	Very many	Few
3	6·0 ,, 1·0	,,	Many	,,
4	6·0 ,, 1·0	Many	,,	,,
5	6·0 ,, 1·2	Very many	Very many	,,
6	6·0 ,, 1·0	,,	,,	,,
7	6·0 ,, 0·8	None	,,	,,
8	6·5 ,, 0·8	,,	Few	None
9	6·5 ,, 1·2	,,	,,	,,
10	7·0 ,, 1·2	Many	Very many	Few
11	7·0 ,, 1·5	Very many	,,	,,
12	7·5 ,, 1·5	Many	,,	,,
13	7·5 ,, 1·2	,,	Many	,,
14	7·5 ,, 1·5	Few	,,	None
15	9·5 ,, 1·5	Very many	Very many	Many

TABLE III.

Fat Solvents only.

	SPLEEN	WEIGHT		
	Size in centimetres	Before treatment	After treatment	
1	4·0 by 0·5	1,760 grm.	2,280 grm.	29 inhalations of benzol
2	4·5 ,, 0·8	1,710 ,,	2,220 ,,	29 ,, ,,
3	5·0 ,, 1·0	1,720 ,,	2,280 ,,	26 ,, ,,
4	5·0 ,, 1·2	1,860 ,,	2,410 ,,	31 ,, ,,

during which they were exposed to the fumes of the gas. Until we have more data it is out of place to draw any conclusion from these tables, but the work seems to corroborate

the suggestion of Dr. Paul Lewis [3] and his associates in the Phipps Institute, that in some animals there is a close association between the spleen and a tuberculous process.

TABLE IV.

Fat Solvents and Tuberculosis.

Rabbit No.	Spleen		Lungs	Kidneys	Treatment
	Size in centimetres	Tubercles	Tubercles	Tubercles	
1	3·2 by 0·5	None	Few	None	11 inhalations of benzol
2	4·5 ,, 0·8	,,	,,	,,	11 ,, ,,
3	5·0 ,, 0·7	,,	,,	,,	11 ,, ,,
4	5·0 ,, 1·0	Few	Many	Few	10 ,, ,,
5	5·2 ,, 1·2	None	None	None	11 ,, ,,
6	5·5 ,, 1·0	,,	Few	,,	10 ,, ,,
7	5·5 ,, 1·3	Few	Many	Many	5 ,, ,,
8	5·0 ,, 1·0	None	None	None	14 ,, ether
9	5·5 ,, 0·7	Few	Very many	Few	10 ,, ,,
10	6·0 ,, 0·5	Many	Many	,,	10 ,, benzol
11	6·0 ,, 1·3	Few	,,	,,	7 ,, ,,
12	6·0 ,, 1·3	,,	,,	,,	7 ,, ,,
13	6·0 ,, 1·2	Very many	Very many	Many	7 ,, ,,
14	6·0 ,, 1·3	None	Many	None	6 ,, ether
15	6·5 ,, 0·5	Very many	,,	Few	10 ,, benzol
16	6·5 ,, 1·8	Many	,,	,,	19 ,, ,,
17	6·5 ,, 1·5	Few	Very many	,,	21 ,, ether
18	6·5 ,, 1·8	,,	,,	None	7 ,, ,,
19	7·0 ,, 1·2	None	Few	,,	8 ,, benzol
20	7·0 ,, 1·5	Very many	Very many	Few	28 ,, ,,
21	7·0 ,, 2·0	None	,,	,,	20 ,, ,,
22	7·0 ,, 1·7	Few	,,	,,	14 ,, ,,
23	7·0 ,, 1·5	Many	Many	Many	5 ,, ,,
24	7·0 ,, 1·3	,,	,,	Few	7 ,, ,,
25	7·0 ,, 1·2	None	Very many	None	8 ,, ether
26	7·0 ,, 1·7	Many	,,	Few	7 ,, ,,
27	7·0 ,, 1·5	Very many	,,	,,	7 ,, ,,
28	7·5 ,, 1·7	Many	,,	,,	1 ,, chloral
29	8·0 ,, 2·3	Very many	,,	Very many	7 ,, benzol
30	8·0 ,, 1·7	,,	,,	Many	6 ,, ,,
31	8·2 ,, 1·5	None	Few	None	10 ,, ,,
32	8·5 ,, 2·0	Very many	Very many	Few	6 ,, ether
33	8·5 ,, 1·3	Many	Many	,,	7 ,, benzol
34	9·0 ,, 1·8	,,	,,	,,	7 ,, ,,
35	9·0 ,, 2·0	Very many	,,	Many	10 ,, ether
36	9·0 ,, 1·8	Many	Very many	Few	7 ,, ,,
37	9·0 ,, 2·0	,,	,,	,,	8 ,, benzol
38	9·5 ,, 1·7	Very many	,,	,,	1 ,, ether
39	13·0 ,, 3·0	,,	,,	Very many	7 ,, benzol

This relation may possibly be associated with conditions that may be further elucidated by a study of such substances as we have used. We chose fat solvents for another purpose, that of dissolving the tubercle bacillus after entrance to the pulmonary stream. In this we were disappointed, as the tuberculous process was made worse instead of relieved.

There is, however, an interesting bio-chemical field open for study (as we suggested in 1912 [4]) in the relation between the substances poured into the inferior vena cava, as represented by the splenic vein contents, and tuberculosis of the lungs, which we trust will come in for more extended research.

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THE ADJUSTMENT OF PRESSURES IN INDUCED PNEUMOTHORAX, WITH ESPECIAL REGARD TO THE MOBILITY OF THE MEDIASTINUM AS INDICATED BY THE MANOMETER.

By C. D. PARFITT, M.D., C.M.

GRAVENHURST, ONTARIO.

During the treatment of phthisis by compression of the lung, there is need of exercising a nice judgment regarding the degree of pressure that should be maintained throughout a long series of injections. Our experience is that the displacement of the mediastinum is of first importance in the adjustment of pressure at any time, and our purpose is to show how this may be estimated with fair accuracy by the aid of the manometer and without the help of the X-ray when the result of physical examination is uncertain.

The first consideration is to obtain a satisfactory collapse of the diseased lung, but while one is trying to effect this a compromise in the pressure desired may have to be made for the safety of the less diseased lung and of the heart. Cases in which collapse of the lung takes place easily seem rarely to suffer strain to either organ if the collapse is gradually induced, and these are the cases in which the mediastinum will usually be found movable.

The cases in which collapse of the lung is more difficult, unless this difficulty is due solely to an induration of the lung, will, because of thickening of the pleura and adhesions, have

a much less easily displaced mediastinum. This relative rigidity of the mediastinum then becomes a great protection to the very cases that require considerable pressure to reduce the size of the lung. With lateral adhesions in the upper part of the thorax a marked displacement of the mediastinum may nevertheless take place.

The effect that the extra work will have upon the functionating lung is a source of continual anxiety. The beneficial influences which, according to accepted theories, account for its improvement, may be sufficient to arrest progress of disease or render it quiescent; but during the long course of treatment indolent progress may be noted after earlier marked improvement. This lung becomes enlarged, emphysematous, and elongated. Lord considers the arguments in favour of the beneficial influence of the overwork of the better lung as invalid. Aron sees little chance for healing of disease in the better lung because of its greater respiratory movement, and finds it impossible to imagine that the better lung can heal under such conditions. There is such danger to a diseased lung from the greater excursion that he urges that on no account should compression be pushed so far that breathing is stopped entirely in the compressed lung. The type of respiration becomes modified as is best suited to the need of the individual at any one time (Brown), and Aron finds in man that the untreated lung breathes with increasing depth the more the treated lung is excluded from respiration, as is shown by greater fluctuations of a manometer connected with the pleural cavity of the functionating lung. Aron's experiments on animals show the concurrent changes of pressure in the pleura of the functionating side, while one lung is gradually excluded from respiration. After a mean pressure of zero has been reached the depth of respiration

which, following an initial increase, has steadily lessened and has been compensated by increased frequency of respiration, promptly increases with lessened frequency of breathing, and at the same time there is a very marked rise in blood-pressure. The lessening depth of respiration of the functionating lung is shown in the pleural cavity by the negative pressure becoming less marked on inspiration and more marked on expiration, thus showing a decreased amplitude.

There may be embarrassment to the circulation with either fixed or movable mediastinum, since, on the one hand, rigidity may cause pressure to be brought to bear upon heart, vessels and nerves, and, on the other hand, flexibility will permit varying tension, kinking, strain and pressure as well.

The right heart, besides the handicap of diminished vascular area in the collapsed or compressed lung and the loss in part of the pump action of the thorax, has to undertake the maintenance of an increased circulation in the functionating lung, and is further embarrassed by more or less lateral displacement (sometimes with a twist on either vertical or horizontal axis, or both), and the pressure of the relatively unyielding gas cavity. Moreover, the heart is the first to feel the effect of diminished oxygenation and the general need of oxygen also causes a rise in pressure in the systemic circulation. Pressure or strain upon nerves may also affect the innervation. The fact that the right heart hypertrophies in both man and animal experiment has been shown at autopsy; but it has also been shown that it sometimes fails to do so. Improvement in the heart's action through lessened toxæmia and improved general condition more than make up for these embarrassments in favourable cases. The heart also may benefit by being enabled to return to a more normal position after previous great displacement due to a contracted lung.

As to a satisfactory collapse of the diseased lung, various writers set different ideal of attainment, though all aim at the exclusion, if possible, of the treated lung from respiration. Saugman and Hansen, while approving Forlanini's objective of compression as a curative measure, were only able to obtain complete compression in 15 per cent. of their cases. On the other hand, Dunham and Rockhill, using the operation as a palliative measure and controlling their work with the X-ray, found that refilling should be frequent and the gas pressure slight, the amount of gas injected being just sufficient to keep the lung collapsed and no more, a zero inspiratory pressure being more than enough. Brauer and Spengler emphasize the general rule that the better and larger the pneumothorax obtained the less need be the pressure, and they consider the object of the operation has been reached as soon as there is no negative inspiratory pressure. When the lung is not too dense, a pressure of 2 to 4 cm. of water is by various writers considered to be sufficient to keep the lung compressed if there are no adhesions. This pressure, if well borne, may be increased to 7 to 8 cm. If adhesions remain, possibly 40 cm. may be necessary (Rist). Saugman has maintained a pressure of 20 to 30 cm. water for months, but has never exceeded 8 cm. Hg. (108·8 cm. H_2O), while Brauer's limit has been 4·5 cm. Hg. Davies considers it useless to persevere in the separation of adhesions when a pressure greater than 1 cm. Hg. (13·6 cm. H_2O) is necessary.

Exclusion of the lung from respiration becomes, from the standpoint of practice, a relative matter, since the frequency of refills necessary to maintain a consistent pressure is scarcely practicable, and in the intervals considerable re-expansion may take place. When the pneumothorax is established an optimum point of pressure is usually found

for each individual, determined for him mainly by the degree of tension the mediastinum will bear.

It has been shown by experiment that those animals bear pneumothorax best in which the elasticity of the mediastinum best resists its displacement (Bruns). The mediastinum is strengthened also by the deposit of fat. Man is fortunate in having one with fair elastic tension, because of the relatively small distance between the heart and the sternum. However, at the site of the atrophied thymus there is a weak area which extends from the second to the fourth cartilage, and may be 3 or 4 cm. deep, filled with loose connective tissue and fat. Another area where the pleura of the right side is relatively unsupported lies between the aorta and œsophagus, between the fifth and twelfth vertebræ (Nitsch). At these situations the pleuræ are capable of further distension after a certain pressure, which first causes a general displacement of the mediastinum, has been passed. There are, apparently, great individual differences in the resistance of the pleuræ to pressure. A healthy pleura will bear a considerable positive pressure from inflation without causing dyspnœa. A recently inflamed pleura becomes relaxed and yields more readily to pressure than the normal pleura, while more chronic intrathoracic inflammation and thickening increase the resistance. Pressure is found to be borne with less dyspnœa when the pleura is thickened than when it is thin. This may be seen in pleural effusion, which, when recent and with a lower pressure, produces greater dyspnœa than when the pleura is thickened and the pressure is higher.

For determining the pressure that will be the best compromise for the individual case, it is, therefore, important to take into consideration the degree of movableness or rigidity of the mediastinum. The index of this is the posi-

tion of the heart, which it may or may not be possible to locate accurately by physical examination. The X-ray is not always available, or it may be impossible to use it at every séance; but without changing the position of the patient the manometer gives information as to mediastinal displacement which serves as a guide at every refill. Naturally, information will be incomplete during the earlier inflations, at least until the zero point has been approached.

v. Muralt, and Hamman and Sloan also, state that the manometer may indicate a too movable or too rigid mediastinum. Their views, however, are completely at variance. v. Muralt says that other things being equal a flexible mediastinum is shown by a rapid rise in pressure with a small respiratory movement of the fluid in the manometer; a rigid mediastinum by a slow rise in pressure with large respiratory movement of the fluid. Hamman and Sloan hold that a slow increase in pressure with wide respiratory variation indicates a flexible mediastinum; a rapid increase in pressure, with small respiratory variations, a rigid mediastinum.

Contrary to these writers, we have found that a flexible mediastinum is shown by a slow rise in pressure with relatively small amplitude of movement of the fluid in the manometer on breathing, and a rigid mediastinum by a more rapid rise in pressure with relatively large amplitude. The slow rise in pressure, when the mediastinum is unduly flexible, is so obvious that it seems probable that v. Muralt has not correctly stated his position. In his paper some ambiguity of the context suggests that this is so. Moreover, with a flexible mediastinum there are frequently to be noticed certain relatively stationary points at which there is no further rise of pressure after one, or even two, decilitres of gas have been introduced. These

points are most frequently noticed when the mean pressure is near zero. The oscillations are also sluggish when the mediastinum is movable. When the mediastinum is more rigid the stationary points are not in evidence and the movement of the fluid in the manometer is more abrupt. These observations, recorded in an earlier paper, are in accord with the following theoretical considerations:—

The position of the mediastinum at any time is accepted as one of equipoise between varying tensions of the two pleural cavities, the resultants of several factors, chiefly the elasticities of the lungs (West). When the tension of one pleural cavity is lessened "the heart, the most delicate index of a change of tension on either side," will move towards the side on which the intrapleural tension is greater, *i.e.*, on which the negative pressure is more marked, "pushed in that direction by a force equal to the differences of those in the two pleural cavities" (Emerson). In expiration, with a general relaxation of the thoracic wall and diaphragm, this position of equilibrium of the mediastinum necessarily exists, and during quiet respiration the same condition obtains since the central tendon of the diaphragm does not stir. During more active inspiratory movement, however, modifying forces come into play so that the position of the mediastinum becomes a resultant of the antagonistic intrapleural tensions and the tension put upon the mediastinum by the combined influence of the descent of the diaphragm and deepening of the thorax. The entrance of gas into one pleural cavity will, however, relax the diaphragm on that side and its dome will become lowered. This permits the mediastinum to yield itself the more readily to satisfy the variable tensions of the two pleural cavities. Irrespective of prevailing intrapleural tensions, inspiration will have the effect of flattening the curves, so to speak, of the irregular

mass forming the mediastinum. This would even be the case in an open elastic cage with bulging central partition if the antero-posterior and vertical diameters were enlarged. This stretching with flattening of the bulge of the mediastinum on inspiration becomes of great importance in open pneumothorax as an aid to the respiration of the sound lung, as pointed out by Sehrwald. Thereby a greater intrapleural tension is created on the sound side, which is a necessary factor for its increased respiration.

The differences of tension between the pleural cavities while a closed pneumothorax is being induced is shown in the tables of Aron's experiments. Though obtained in animals analogous results would be expected in man. As might be anticipated, the actual differences in tension between the two cavities at each phase of respiration shows a constant increase in tension in favour of the pleural cavity of the functionating lung. Except for a very short time early in the operation this difference of tension is much greater on inspiration. If relative intrapleural tension alone adjusts the position of the mediastinum, it would at first sight seem that the mediastinum should move towards the sound side on inspiration and away from it on expiration. That the reverse movement obtains is shown by the fluoroscope and skiagram (figs. 1 and 2), and on inspiration the mediastinum is seen to move towards the pneumothorax cavity and away from it on expiration. The degree of this movement will necessarily depend upon the relative mobility of the mediastinum. If flexible, the movement is much more marked when there is considerable gas in the cavity than when it is relatively empty. In the latter instance, a more normal condition is approached, since the diaphragm is less relaxed, the intrapleural tension is increased, and there is less necessity for a readjustment

of the position of the mediastinum. The explanation of the movement of the mediastinum toward the pneumothorax cavity given by Bittorf, and Saugman and Hansen also, is that the inspiratory increase of tension in the pneumothorax cavity causes the mediastinum to move towards that cavity. Because of the experiments just mentioned this explanation demands consideration, since there is a relatively greater tension on inspiration in the pleural cavity of the functionating lung. The enlarged thorax on expiration will, however, cause a shift of the mediastinum to the pneumothorax side through the altered intrathoracic pressure, modified, of course, by the restraining opposed tension of the sound pleural cavity. The values of the pressures recorded in the pleural cavity of the functionating side must be accepted with some reservation, since on this side the force of cohesion is practically unimpaired, and this force, according to Brauer and Roth, abolishes the negative pressure which becomes a fact only after a pneumothorax has arisen.

The mediastinum, then, in an artificial pneumothorax, may play an active part on inspiration, and a passive one on expiration, when its position is determined by the opposing intrapleural tensions.

Therefore, when the mediastinum is movable the inspiratory enlargement of the thoracic space on the side of the pneumothorax will be reduced by the encroachment of the mediastinum upon it, which is made possible by the inrush of air into the untreated lung, and this yielding of the inner wall of the cavity will in part neutralize the increased intrathoracic tension on the treated side. At the same time the partially collapsed lung will have occasion to expand less than if the inner wall were more rigid, and with the force of cohesion lost it is not in such good con-

dition to expand as is the functionating lung. Because of its greater elasticity, the functionating lung will also yield more readily on expiration than will the partially collapsed lung, the mediastinum will move to that side and room will so be found for the gas cavity, which will have its tension somewhat increased and pressure reduced by the greater tension of the sound pleura transmitted to it.

With the effect of inspiration and expiration upon the gas in the pleural cavity partially neutralized by this swaying of the mediastinum, the pressures registered in the manometer will be less than if the full force of the expansion and contraction of the chest wall had obtained, and the amplitude of movement of the fluid will consequently be less. (Note the especially small amplitude in Chart I.)

When the mediastinum is rigid it will take less advantage of the relaxation of the diaphragm on the introduction of gas to yield to the opposing intrapleural tensions. On inspiration it will be but little modified in form and will, therefore, not neutralize the effect of the enlargement of the pneumothorax cavity by the enlargement of the chest wall and the descent of the diaphragm. The respiratory movement will then more markedly influence the expansion of the collapsed lung, but simultaneously there will be more marked inspiratory and expiratory pressures recorded by the manometer. The amplitude will consequently be greater. (Contrast amplitude of B with C and D, Chart II.) The amplitude in either case is, however, subject to modification in degree because of the varied conditions of breathing which best suit the patient's need at the moment. Emerson has shown that the chest may assume different sizes on the introduction of fluid, while the intrathoracic tension remains approximately the same, and that the respiratory oscillations are not in direct proportion to the respiratory movements,

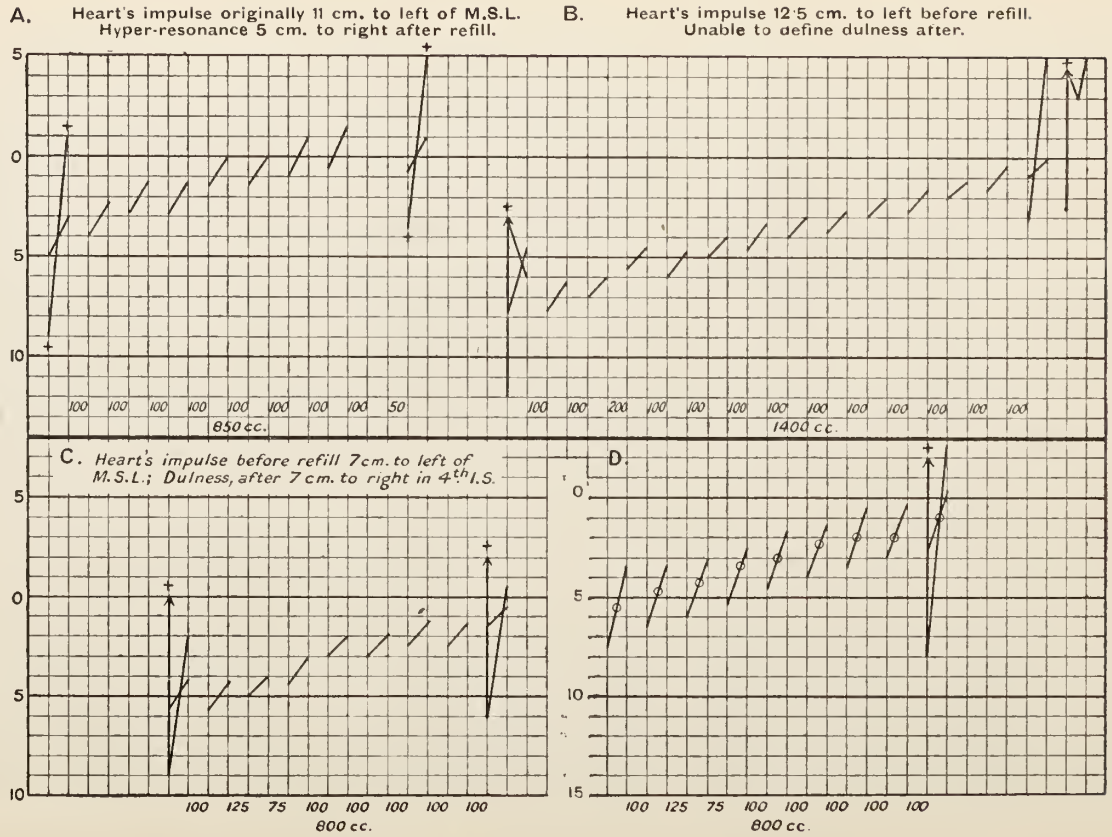
A. Heart's impulse originally 11 cm. to left of M.S.L.
Hyper-resonance 5 cm. to right after refill.
B. Heart's impulse 12·5 cm. to left before refill.
Unable to define dulness after.
5
0
5
10
100 100 100 100 100 100 100 100 50
850 cc.
100 100 200 100 100 100 100 100 100 100 100 100 100
1400 cc.
C. Heart's impulse before refill 7 cm. to left of M.S.L.; Dulness, after 7 cm. to right in 4th I.S.
5
0
5
10
100 125 75 100 100 100 100 100
800 cc.
D.
0
5
10
15
100 125 75 100 100 100 100 100
800 cc.

Chart I, Case 1. Movable mediastinum. (See figs. 1 and 2.)

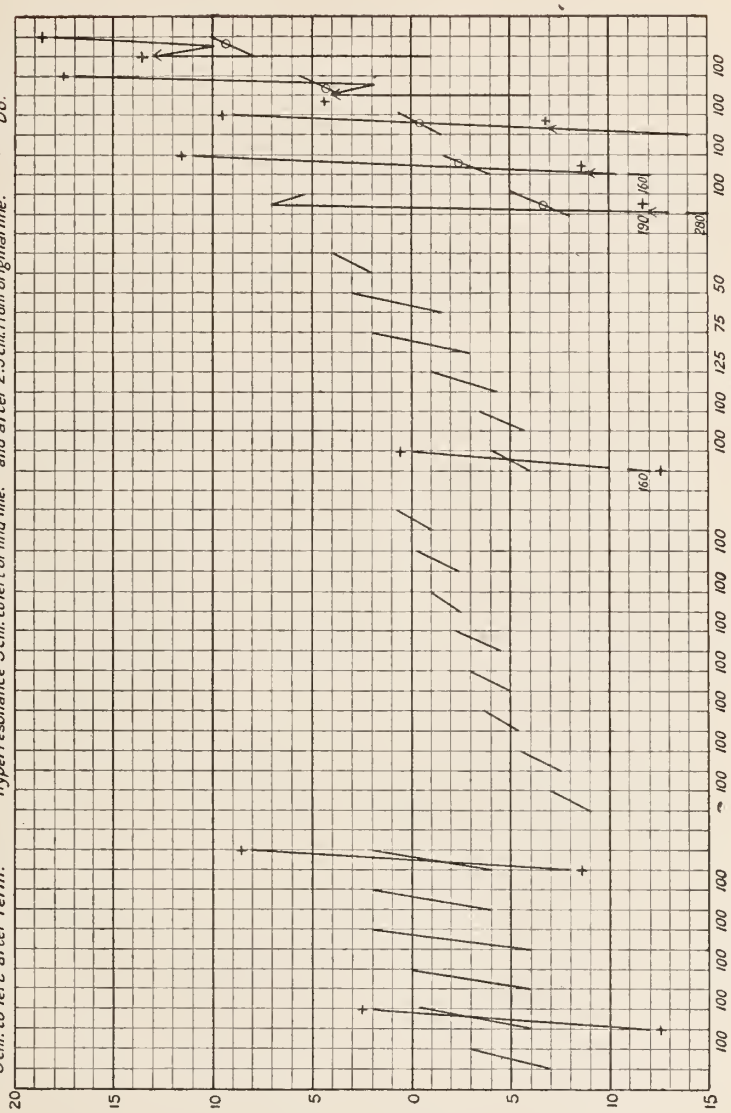

Chart II, Case 2. Movable mediastinum becoming fixed.

the efficiency of which bears an inverse relation to the degree of distension.

In either case, also, the pressures will be modified by the degree of expansion of the treated lung and some confusion in interpretation may be caused. As pointed out by Hamman and Sloan, a re-expanded lung is shown by a very gradual rise in pressure on introducing each decilitre of gas, while a lung that remains collapsed is shown by a slow rise at first, and then a very sudden and marked rise. The stationary points already mentioned, at which the pressure fails to rise following the introduction of one or two consecutive decilitres of gas, help, we think, to distinguish such conditions. (See A, C, D, Chart I, and A and B, Chart II.) They are points of emphasis in the curve characteristic of the slow rise of pressure found when the mediastinum is movable. They may, perhaps, be explained by a somewhat irregular and momentarily more marked satisfaction of the elasticity of the functionating lung, the gradual satisfaction of which mainly accounts for the relatively slow rise in pressure, made possible by the further yielding of the mediastinum, partly the result of the continued relaxation of the diaphragm, rather than by an enlargement of the pleural space caused by the irregular enlargement of the thoracic wall, or than by an irregular collapse of the treated lung. We may surmise that the weak areas described by Nitsch play some part in their causation, as it is not unusual for hyper-resonance to extend markedly to either side of the sternum, even without a positive intrapleural pressure. High levels of the stationary points, which may have been caused by bulging of such weak areas, were found in a case of very large pneumothorax, with extreme movableness of the mediastinum. At any rate, stationary points are found in consecutive refills to be more or less constant at some

one or at several levels in the pressure curves in cases in which the mediastinum is readily displaced, and are not found in those in which it is relatively fixed. (See Charts I and II.)

The pressures for deep respiration, taken both at the beginning and the end of the refill, give information which is also suggestive and point to either condition, although the risk of rupture of the lung makes such deep breathing a dangerous experiment. With flexible mediastinum the amplitude for deep respiration is not disproportionate to that for quiet respiration (see Chart I and Chart II, A), and is about the same at the end of the refill as at the beginning. The incompletely collapsed lung no doubt plays a material part here as well as the yielding mid-wall. With rigid mediastinum, and especially if the rigidity of the pneumothorax wall is, so to speak, increased by a lung which does not readily re-expand, the amplitude may be quite disproportionate to that for quiet respiration, and extreme (see D, Chart II) both at the beginning and at the end of the refill. There may also be material differences in the amplitude before and after the inflation depending upon the recompression of the lung and upon the muscular power that is brought to bear. When a rigid mediastinum obtains and the amplitude for forced respiration is relatively small, it is probable that there is considerable lung excursion which compensates the respiratory movements.

Needles of relatively small bore are, we think, better adapted for the estimation of varying amplitudes of movement of the water column than larger needles. With the latter the slowly rising curve and the stationary points can only be well shown by determining the mean pressure, after the manner adopted by Saugman, by means of a valve on the rubber connection between the limbs of the manometer,

or by a valve on the tubing to the manometer, as has been suggested by the writer. The device recommended by v. Adelung for making mean readings by means of a by-pass might be modified so as to give a suitable standard calibre for noting oscillations, and this would make one independent of variations in the bore of different needles. The pressures recorded through a small needle will lie in the upper half of the amplitude recorded by a large needle, *i.e.*, near the true mean pressure shown with a large needle (see Chart I, C and D). Needles of a bore of ·018 in. and ·022 in., B.W.G. 26 and 24, have been mainly used. An amplitude of 2 cm. or less with a needle of ·018 in. bore is suggestive of mediastinal displacement, and the more the excursion decreases or increases from this measurement, the more or less suggestive it becomes.

We have also previously described a phenomenon, not infrequently found, which we have not seen mentioned elsewhere, but it is of some interest here. When the patient is asked to hold the breath at the end of inspiration, the level of the fluid in the manometer suddenly rises several centimetres (see Chart I, B, C, D; Chart II, D). At the end of the rise, when he is asked to let out the breath, there is a momentary drop in the level of fluid of about a centimetre; the pressure then rises again with continued expiration to a point dependent upon conditions already described. This phenomenon was noted in six out of fifteen cases with large gas cavities, and in three out of fifteen cases with small gas cavities. It occurs with ordinary respiration as well as with forced respiration, is usually present both at the beginning and end of the séance, and it seems to be largely independent of the degree of pressure. It may be present at the initial and early operations, and then disappear entirely or return some months later; also, when not present at first it may appear later (Charts I and II). It is present

with both types of mediastinum. When this phenomenon was first noted it was confusing, as it was feared that a re-expanded lung had been pierced; but it was invariably noted that the pressure was maintained at the end of the rise and on expiration. The importance of the phenomenon is that it makes an exception to the rule that when the needle is in the pleural space the pressure is maintained on holding the breath at the end of inspiration.

One case (Case 1) with markedly movable mediastinum (figs. 1 and 2) in which this phenomenon is present we had opportunity of examining with the fluoroscope, and a slight jerky rise of the diaphragm was noticed immediately on holding inspiration. This was seen both before and after a refill, at which the beginning and ending mean pressures were −60 mm. and 0 respectively, and at which 1,400 cm. gas had been introduced. Eight skiagrams taken of this patient's chest at different times failed to show diaphragmatic adhesions, though there was a lateral adhesion about the fourth rib. Three other cases in which it is present, with moderately large gas cavities, and each having a fairly rigid mediastinum, showed on plates some diaphragmatic adhesions as well as lateral adhesions.

It is probable that this phenomenon is caused by a contraction of the lower ribs and abdominal muscles when the breath is held on inspiration, with a resulting increase of abdominal pressure and a passive rise of the relaxed diaphragm. With beginning expiration these muscles are first of all relaxed, and this relaxation allows a momentary descent of the diaphragm, with a resulting slight fall in intrathoracic pressure. Bittorf's and Teske's experiments and observations of paradoxical movements of the diaphragm are in accord with this explanation.

The practical application of these inferences may be seen from the following typical cases and accompanying charts :—

Case 1.—Freely movable mediastinum. (Chart I. Figs. 1 and 2.) No. 23, female. Collapse of left lung for hæmorrhage recurrent over a period of five years. Left lung: disseminated fibroid disease throughout, not of intense character. Right lung: slight infiltration above third rib.

June 23, 1914.—Initial reading (large bore needle, ·042 in., 19 B.W.G.), −96 to −80 mm., deep respiration, −140 to −20, 200 c.c. air, ending middle pressure 0. A network of adhesions was present. The phenomenon was noted. A fairly large pneumothorax was readily obtained, which ultimately required an average of 1,000 c.c. gas every three to four weeks. Breath-sounds were almost completely suppressed after the inflation. After the fourth inflation there was no sense of pressure, but the patient felt very tired for about a week after each refill. Dyspnœa was felt after a mean positive pressure of 5 to 10 mm. Hyper-resonance was 5 cm. to the right of the mid-sternal line. The apex beat, which before the initial inflation was felt 11 cm. to the left of the mid-sternal line, and which could usually be felt in almost the same position before a refill, became lost after one. Pulsation was occasionally seen after a refill 5 cm. to the right of the mid-sternal line, and relative cardiac dulness noted 5 to 7 cm. to the right. The characteristic curve of a movable mediastinum was early noted (Chart I), and has been consistently maintained. The deep respirations have never been disproportionate to those of quiet breathing, and their relative values have always been consistently maintained. Because of this feature and the subjective symptoms the mean pressure was reduced to 0, or a little less. This reduction lessened the sense of fatigue somewhat, and the pulse, which had averaged 90, fell to a steady rate of 80 after two weeks. The sputum, small in amount, was but little influenced. The physical signs in the right upper lobe have diminished somewhat. The general condition of the patient has materially improved, and apprehension has gone. Skiagrams (figs. 1 and 2) show a marked displacement of the heart after the refill, which is materially more marked on expiration than on inspiration. There is a lateral adhesion seen about the fourth rib, but no diaphragmatic adhesion. The fluoroscope shows the greatest movement of the heart with respiration when there is a large amount of gas. The lung is not completely collapsed immediately after a refill.

FIG. 1.—Full inspiration.

FIG. 2.—Full expiration.

Movable Mediastinum.—Skiagrams of chest taken third day after refill of April 3, 1915. See Case 1, Chart I.

The yielding of the mediastinum, therefore, makes complete collapse a doubtful possibility or possible only at considerable risk. The phenomenon was present during the first five fillings, and then disappeared until the ninth filling, since which time it has been usually present (see Chart I, B, C, D,). Through the fluoroscope the diaphragm was seen to rise in a jerky manner when the breath was held on inspiration.

Case 2. — Movable mediastinum becoming fixed. (Chart II.) No. 27, female. Right lung collapsed for severe and long-standing disease. Left lung: slight disseminated disease throughout upper lobe.

September 21, 1914.—Initial operation, adhesions present, 200 c.c. gas. Two days later gas easily introduced, two sets of stationary points noted (Chart II, A) with the mean pressures at approximately −20 and −5 mm., large oscillations with large bore needle ·035 in. (B.W.G. 20). The further giving way of adhesions may account for these stationary points; gas, 500 c.c.; apex beat shifts, 2 cm.; no material difference between the extremes of deep inspiration and expiration at the beginning and end of the operation. Since the second inflation breath sounds immediately after the operation have been almost completely suppressed. By December 8, seventh refill, a large pneumothorax has been obtained. Hyper-resonance extends 3 cm. to the left of the mid-sternal line; the left margin of relative cardiac dulness and the apex beat have each been displaced 4 cm. by the inflation. Curves with stationary points have been consistently seen. The rise is fairly slow; the oscillations rather irregular (Chart II, B). January 2: The rise in pressure for a smaller amount of gas, 500 c.c., is more abrupt, and the oscillations are greater. Two days later the patient suffered great pain at the base of the left lung, which disappeared suddenly after four days. January 30: The rise in pressure is more rapid (*c*, Chart II), from −50 to + 30 mm. by 450 c.c. gas. No stationary points are noted; the oscillations are materially larger but variable; suggestion of fluid in the needle; the excursion on deep respiration has greatly increased. The relative cardiac dulness is fixed at 8·5 cm. to left of the mid-sternal line both before and after the inflation. The characteristics of a fixed mediastinum are now seen, and these continue. The succussion sign of fluid was obtained after the tenth refill, but was not found again. At the twelfth refill the deep respiratory excursion became

enormous, showing an amplitude of 230 mm. and 220 mm. before and after the inflation (Chart II, D). The phenomenon was first seen at the end of this inflation, but has since been constant. It is well illustrated in this chart (Chart II). Since the mediastinum has become fixed the pressures have been consistently raised, so that now the ending mean pressure is 90 mm. The patient is more comfortable, with almost entire absence of expectoration, and physically better than at any time since the pneumothorax was induced.

The first case illustrates the correlation between fatigue symptoms, physical signs of displacement of the mediastinum, and the data here given for inferring a movable mediastinum from the readings of the manometer. Relief from the symptoms was obtained by reducing the pressure to just below zero. The second case required care in the adjustment of pressure for both the functionating lung and heart. Restraint was exercised in the pressure used, and frequent refills were undertaken until the readily displaced mediastinum became fixed, following the pleurisy, which fixation is well shown by the manometer. It then proved to be beneficial to increase the pressure, and after this the intervals could be lengthened. A number of other cases might be cited to show the correlation between physical signs and the manometric indications of mediastinal displacement. In some cases in which the physical signs of displacement failed or were less marked, the manometer has proved a distinct help in estimating the condition of the mediastinum.

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EXPLANATION OF CHARTS.

Amplitude is shown by length of line; the shorter lines quiet, the longer, deep breathing.

Inspiration, the lower end; expiration, the upper end.

The circle on the amplitude line shows the mean pressure.

+ Signifies that the level of the fluid is maintained when the breath is held.

The arrow shows the phenomenon of rise of pressure at end of inspiration.

CHART I (Case 1).—Movable mediastinum (*see* figs. 1 and 2).

(*a*) Sixth refill, August 12, 1914. Interval twenty-four days. Bore of needle, ·018 in.

(*b*) Seventeenth refill, May 27, 1915. Interval twenty-seven days. Same needle.

(*c*) Fifteenth refill, April 3, 1915. Interval twenty-nine days. Same needle. Skiagrams taken after this refill.

(*d*) Same refill. Large needle, bore ·042 in. Reading simultaneous with that of (*c*).

CHART II (Case 2).—Movable mediastinum becoming fixed.

(*a*) Second refill, September 23, 1914. Interval two days. Bore of needle, ·035 in.

(*b*) Seventh refill, December 8, 1914. Interval twenty-one days. Bore of needle, ·018 in.

(*c*) Ninth refill, January 30, 1915. Interval twenty days. Same needle.

(*d*) Thirteenth refill, May 11, 1915. Interval twenty-eight days. Same needle.

OBSERVATIONS ON THE BLOOD-PRESSURE IN CASES OF DYSTHYROIDISM.

By JOHN M. SWAN, M.D.
ROCHESTER, N.Y.

In this paper I have brought together the blood-pressure observations made in fifty cases of dysthyroidism of varying degrees of intensity. The cases have been analysed from the point of view of systolic blood-pressure, diastolic blood-pressure, and pulse-pressure. I have also studied the various factors concerned in determining the functional capacity of the heart as described in the paper which I presented at the meeting of this Association in 1914[1]. I also include a single observation of the alterations in blood-pressure during and after a hemithyroidectomy. Observations were made every five minutes during the operation, and after the operation other observations were made at varying intervals for several weeks.

The blood-pressure observations were made with a Riva Rocci instrument, except those marked *, which were made with a Tycos instrument. A 12 cm. cuff was used.

In a paper on the influence of carbonated brine baths on blood-pressure [2] I adopted the figures of Woley [3] as normal; and in this paper I shall use the same standard for comparison. In 1,000 estimations of blood-pressure, in supposedly healthy individuals, Woley found that the average

systolic pressure of persons from 15 to 30 years of age was 122 mm.; of persons from 30 to 40 years of age was 127 mm.; of persons from 40 to 50 years of age was 130 mm.; and of persons from 50 to 60 years of age was 132 mm. I have purposely chosen the averages for comparison rather than the average high or average low pressure.

TABLE I.—NORMAL SYSTOLIC PRESSURE.

Case	Sex	Age	Systolic	Diastolic	Pulse	C. E.	Overload	Per cent., second phase	C. S. : C.W. ratio	Time interval
						Per cent.	Per cent.			
*107*a*	F.	49	130	80	50	38·0	12·0	16·0	56·0 : 44·0	—
			148	90	58	39·0	14·0	10·3	—	6 days
			164	104	60	36·0	7·0	20·0	—	8 months
			170	104	66	38·0	13·0	12·1	—	15 months
127*d*	F.	28	120	60	60	50·0	—	10·0	60·0 : 39·9	—
*178*a*	F.	30	118	70	48	40·0	18·0	4·1	70·7 : 29·1	—
187	M.	27	126	79	47	37 0	9·0	59·5	82 9 : 16·9	—
			*126	72	54	42·0	25·0	51·8	66·6 : 33·3	5 months
			*160	74	86	53·0	66·0	23·2	53·4 : 46·4	3 days
			*144	78	66	45·0	34·0	54·5	66·6 : 33·3	24 hours
			*142	60	82	56·0	86·0	26·8	41·4 : 58·4	24 hours
			*140	60	80	57·0	83·0	30·0	62·5 : 37·5	24 hours
			*130	54	76	58 0	90·0	28·9	52·5 : 47·3	After dressing wound
			*132	64	68	51·0	56·0	26·4	41·1 : 58·7	24 hours
			*124	74	50	40·0	17·0	32·0	48·0 : 52·0	2 days
			*128	70	58	45·0	21·0	20·6	55·0 : 44·7	After dressing wound
			*130	64	66	50·0	53·0	30·3	45·4 : 54·5	2 days
			*128	64	58	47·0	40·0	51·7	72·3 : 27·4	2 days
			*132	60	72	54·0	70·0	44·4	61·0 : 38·8	4 days
			*136	64	72	54·0	62·0	36·2	61·2 : 38·8	2 days
			*134	84	50	37·0	9·0	60·0	80·0 : 20·0	11 days
			*128	62	66	48·0	56·0	—	—	1 week
			127	90	37	29·0	−9·0	48·6	59·4 : 40·5	22 months
299	F.	40	124	75	49	39 0	15 0	38·7	79·5 : 20·4	—
			129	80	49	38·0	11 0	51·0	—	9 months
			146	80	66	45·0	32·0	10·6	—	13 months
			120	70	50	41·0	21·0	20·0	—	17 days
			124	80	44	35·0	5·0	29·5	—	2 months
			118	78	40	33·0	1·0	22 5	82·5 : 17·5	1 month
331	F.	35	123	64	59	48·0	42·0	13·5	55·8 : 43·9	—
			85	47	38	44·0	30 0	39 4	65·7 : 34·1	10 months
			105	55	50	47·0	40·0	50·0	—	38 days
			120	68	52	43 0	26·0	—	—	3 months

TABLE I.—NORMAL SYSTOLIC PRESSURE (*continued*).

Case	Sex	Age	Systolic	Diastolic	Pulse	C. E.	Overload	Per cent. second phase	C. S. : C.W. ratio	Time interval
						Per cent.	Per cent.			
383	F.	38	125	76	49	39·0	14·0	—	—	—
			121	70	51	42·0	22·0	9·8	64·7 : 35·2	16 months
389	M.	32	125	80	45	36·0	6·0	8·8	64·3 : 35·5	—
			128	69	59	46·0	35·0	25·4	81·1 : 18·5	14 months
			150	75	75	50·0	50·0	24·0	76·0 : 23·9	3 months
438	M.	24	120	79	41	34·0	1·0	—	—	—
476	M.	32	127	95	32	25·0	−17·0	15·6	—	—
486	F.	28	126	85	41	32·0	−2·0	19·5	—	—
			105	74	31	29·0	−9·0	48·3	—	4 months
			120	71	49	40·0	19·0	18·3	48·9 : 50·9	2 months
488	M.	33	125	70	55	44·0	28·0	50·9	74·5 : 25·3	—
499	F.	38	135	85	50	39·0	8·0	—	—	—
			*134	80	54	40·0	17·0	—	—	2½ months
503	F.	56	140	90	50	35·0	5·0	—	—	—
518	M.	58	125	65	60	48·0	42·0	16·6	—	—
519	M.	26	122	80	42	34·0	2·0	—	—	—
*520	F.	24	126	68	58	45·0	35·0	17·2	86·1 : 13·7	—
†529	F.	38	128	63	65	50·0	53·0	35·3	46·0 : 53·7	—
			*148	90	58	39·0	14·0	44·8	68·9 : 30·1	1 month
			139	60	79	56·0	81·0	12·6	50·4 : 49·3	7 weeks
			*126	78	48	38 0	11·0	33·3	62·4 : 37·4	1 month
			131	74	57	43·0	27·0	26·3	64·8 : 35·0	2 months
537	M.	50	134	87	47	34·0	4·0	—	—	—
573	F.	25	120	85	35	29·0	−9·0	28·5	71·3 : 28·4	—
			*128	80	48	37·0	10·0	—	—	25 days
			*126	76	50	31·0	15·0	32·0	80·0 : 20·0	1 week
			*126	90	36	28·0	−10·0	27·7	—	1 week
			*132	90	42	31·0	−4·0	—	—	1 week
			*130	90	40	30·0	−6·0	—	—	1 week
			97	65	32	32·0	−1·0	15·6	—	1 week
			128	80	48	37·0	10·0	8·3	—	10 days
			105	65	40	38·0	11·0	22·5	—	2 weeks
			90	50	40	44·0	30·0	25·0	45·0 : 55·0	3 weeks
			100	62	38	38·0	11·0	21·0	71·0 : 28·8	4 months
576	F.	30	120	79	41	34·0	1·0	—	—	—

† See special observations on blood-pressure made during and immediately after hemithyroidectomy.

One would expect to find a low systolic blood-pressure in cases of dysthyroidism, because Sanford and Blackford [4] found a marked fall in the systolic blood-pressure in the dog after the intravenous injection of extracts of strumas from cases of exophthalmic goitre. They also found that the serum of patients suffering from acute exophthalmic goitre

produced a marked fall in the systolic blood-pressure. They found that one injection of the depressor substance found in the saline extract of the struma produced a tolerance against subequent injections. They think that the same depressor substance which exists in the saline extract of the struma is present in the blood serum of the patients suffering from the disease.

Fawcett, Rogers, Rahe, and Beebe [5] experimented with watery extracts of fresh thyroid glands of pigs. They have also used the alcohol soluble residue and the alcohol insoluble residue. Injections were made intravenously. The alcohol soluble residue was the only substance that produced appreciable effect on the blood-pressure in their hands. In their experiments no tolerance was produced.

Klose [6] succeeded in producing typical Basedow's disease in dogs by the injection of the fresh juice from a Basedow struma. He found a fall of 20 mm. in the systolic blood-pressure. Devic and Gardere [7] refer to some observations made by Hascovec, who demonstrated a reduction of the systolic blood-pressure after intravenous injections of thyroid fluid.

Upon the basis of Woley's figures, twenty-one of the cases in this series had a systolic blood-pressure, which was normal for the age of the patients; fifteen cases had a low systolic blood-pressure; and fourteen cases had a high systolic pressure.

I.—Normal Systolic Pressure Cases.

The cases in which, at the first examination, the systolic blood-pressure was normal for the age of the patient are 107*a*, 127*d*, 178*a*, 187, 299, 331, 383, 389, 438, 476, 486, 488, 499, 503, 518, 519, 520, 529, 537, 573, and 576 (Table I). Of

these cases ten showed, clinically, a more or less well-defined cardiac hypertrophy, one showed ventricular extrasystoles, and one presented pulsus bigeminus in addition to the cardiac hypertrophy.

In view of the fact that these cases presented cardiac hypertrophy, perhaps they should be classed with the low-pressure cases; because, other things being equal, a cardiac hypertrophy is attended with an increase of the systolic pressure. Of these the pulse-pressure was above 40 in ten; 40 or below in one; although in two the pulse-pressure was 41 only. The cardiac efficiency factor was above 35 in six, between 25 and 35 in five. The overload factor was above 40 in three, and gave a negative figure in two. The percentage of the pulse-pressure formed by the second phase was below 40, or could not be obtained in all.

Case 331 was a female, aged 35 years. Ten months after the first examination was made her systolic pressure registered 85 mm.; pulse-pressure, 38; cardiac efficiency, 44 per cent.; overload, 30 per cent.; and the second phase formed 39·4 per cent. of the pulse-pressure. The C.S., C.W. ratio was as 65·7 : 34·1. This drop in systolic pressure was noted during a relapse after symptomatic improvement. Five weeks later, after three weeks' rest in bed and two weeks living out of doors, her systolic pressure had risen to 105 mm., and three months later had returned to 120. The pulse-pressure in this patient, which was originally 59 mm., fell with the relapse to 38, and then increased to 50 mm. and 52 mm. The cardiac efficiency was always above 40 per cent. The second phase, which at first formed 13 per cent. of the pulse-pressure, formed 50 per cent. at the third examination, but could not be obtained at the last.

Case 573 was a female, aged 25 years. Her systolic pressure was fairly constant, 120 to 132 mm., except for three drops, to 97 mm., 105 mm., and 90 mm. The first drop occurred when, after a period of four weeks' rest in bed, she was allowed to get up, and was started on mild gymnastics. The second was coincident with two attacks

of palpitation, an acute tonsillitis, and constipation. The third without attending phenomena. The pulse-pressure was usually about 40 mm.; on two occasions it was 48 mm., and on one 50 mm. The cardiac efficiency factor was within normal limits six times, in spite of a demonstrable hypertrophy, and above 35 per cent. on four occasions. The second phase was always below 40 per cent. of the pulse-pressure.

Ten of the cases presented no clinical evidence of circulatory disturbance. The pulse-pressure was above 40 in nine of these 40 or below in one, although in two the pulse-pressure was 41 and 42 only. The cardiac efficiency factor was above 35 in seven of these; between 25 and 35 in three (the same cases in which the pulse-pressure was normal). The overload factor was not significant except in one case (476), in which it was −17. The percentage of the pulse-pressure formed by the second phase was below 40, or could not be obtained in eight cases, and was above 40 in two. These figures would seem to indicate that even with a normal systolic blood-pressure these cases showed evidences of functional disturbance of the myocardium.

In *Case* 107*a* a normal systolic blood-pressure at the first examination was converted into a high pressure in six days. Then the pulse-pressure had increased, the cardiac efficiency factor had increased, and the second phase could not be obtained. Eight months later, after a hemithyroidectomy had been done by Dr. Tinker at Ithaca, the systolic, the diastolic, and the pulse-pressures were still higher, the cardiac efficiency factor was nearer the high normal, and the second phase had increased to 20 per cent. of the pulse-pressure. Perhaps this patient belonged to the high-pressure cases originally, and the low systolic pressure at the time of the first examination was due to the effects of an acute infection which was active at the time of the examination.

Case 178 had a normal systolic blood-pressure before his operation. Immediately after the operation, however, the systolic pressure increase to 160 mm.; the diastolic pressure remained at about the same point; the pulse-pressure increased to 86 mm. The cardiac efficiency factor, which before the operation had been 42 per cent., had increased to 53 per cent.; the overload was 66 per cent., and the second phase, which had been 51·8 per cent. of the pulse-pressure before the operation, had decreased to 23·2 per cent. By the time the patient left the hospital, however, the systolic pressure had returned to 128 mm.; the diastolic to 62 mm.; the pulse-pressure to 66 mm. The cardiac efficiency factor was still 48 per cent.; the overload 56 per cent., and the percentage of the pulse-pressure formed by the second phase was not obtainable. This patient was examined one year and ten months after his operation, and his blood-pressure was quite normal. Systolic, 127; diastolic, 90; pulse-pressure, 37; cardiac efficiency, 29 per cent.; load, −9 per cent.; second phase, 48·6 per cent.; C.S., C.W., 59·4 : 40·5.

One might conclude from this case that the blood-pressure disturbance was the direct result of the thyroid secretion, and that the alterations in the myocardial factors were due to a functional disturbance.

In the cases, then, with a normal systolic pressure at the first examination the pulse-pressure was, at a rule, above 40—eighteen out of twenty-one cases. The cardiac efficiency factor was above the normal in thirteen; within normal limits in eight. The overload factor appears not to be excessive. The percentage of the pulse-pressure formed by the second phase is, as a rule, below 40, or cannot be obtained—nineteen out of twenty-one cases. The cardiac efficiency factor is usually disturbed, whether or not clinically demonstrable organic disturbances exist in the circulation.

II.—Low Pressure Cases.

The following cases were cases in which the systolic blood-pressure was below normal at the first examination

TABLE II.—LOW SYSTOLIC PRESSURE.

Case	Sex	Age	Systolic	Diastolic	Pulse	C. E.	Overload	Per cent., second phase	C. S. : C. W. ratio	Time interval
						Per cent.	Per cent.			
180*b*	F.	25	102	65	37	36·0	6·0	—	—	—
266*a*	F.	35	105	65	40	38 0	11·0	—	—	—
312*a*	M.	34	100	67	33	33·0	−1 0	36·3	66·6 : 33·4	—
386*a*	F.	24	114	95	19	16·0	−30·0	—	—	—
430*a*	M.	33	115	68	47	40·0	19·0	—	—	—
483	F.	42	106	66	40	37·0	10·0	27·5	77·5 : 22·5	—
502	F.	20	82	48	34	41·0	20·0	—	—	—
509	F.	30	100	65	35	35·0	3·0	—	—	—
512	F.	39	102	73	29	28·0	−11·0	24·1	65·4 : 34·4	—
			100	60	40	40·0	16·0	15 0	—	6 months
543*a*	M.	20	112	65	47	41·0	22·0	—	—	—
548	M.	37	100	75	25	25·0	−17·0	40 0	—	—
572	F.	36	115	65	50	43·0	26·0	—	—	—
579	M.	26	109	67	42	38·0	12·0	45·2	76·1 : 23·8	—
580	F.	43	109	72	37	33·0	1·0	—	—	—
639	F.	49	125	84	41	32·0	−2·0	—	—	—

(Table II); 180*b*, 266*a*, 312*a*, 386*a*, 430*a*, 483, 502, 509, 512, 543*a*, 548, 572, 579, 580, and 639. Cases 312*a*, 483, 509, 548, 580, and 639 had a clinically demonstrable hypertrophy of the heart.

Of the cases without a clinically demonstrable cardiac hypertrophy the pulse-pressure was below 40 in four, and 40 or above in five. The cardiac efficiency factor was between 25 and 35 in one. It is necessary to say that of the cases in which the cardiac efficiency was above 35, one gave a factor of 36 and two of 38. If we omit these very slight percentages above normal, we still have four of these cases without demonstrable cardiac hypertrophy and with low systolic pressure in which the cardiac efficiency factor is beyond normal, and it would seem that even in such cases there is some definite disturbance of the myocardial function. Perhaps the variation in the pulse-rate between the recumbent and the erect posture ought to have been studied in connection with these findings; but it appears to me that the

cardiac efficiency factor is more dependable as an indicator of myocardial insufficiency than postural variations of the pulse-rate.

The overload in these cases is not significant. The maximum overload was 26 per cent.; the minimum minus 30 per cent. I do not know that I have selected a suitable phrase for representing a cardiac load which is below 50 per cent.; but it seems practicable to refer to the load as a minus factor when it is below the normal of 50 per cent.

In eight of these cases the second phase of the auscultatory blood-pressure determination was below 40, or not obtainable. It was 40 or above in one case. This factor also indicates that in these cases there was a definite cardiac muscle disturbance.

When the C.S. : C.W. ratio was obtainable it was always on the side of cardiac strength; in one case 65·4 : 34·4, in another case 76·1 : 23·8.

Of the cases with a clinically demonstrable cardiac hypertrophy the pulse-pressure was below 40 in four, and 40 or above in two. The cardiac efficiency was between 25 and 35 in five, and above 35 in one. It would seem as though in these cases the hypertrophied muscle had produced a better myocardial efficiency. The overload in these cases was insignificant. The second phase was 36·3 per cent., 27·5 per cent., and 40 per cent. respectively in three cases. In the other three cases it was impossible to obtain the second phase percentage. In the two cases in which it could be worked out the cardiac strength was in excess of the cardiac weakness factor—77·5 : 22·5 and 66·6 : 33·3.

In the cases in which the systolic pressure was below normal there seems to be some difference depending upon the presence or absence of a clinically demonstrable cardiac hypertrophy, not so much in respect to the pulse-pressure as

in respect to the cardiac efficiency factor. In the cases in which no cardiac hypertrophy could be determined the cardiac efficiency factor was above 35 per cent. in eight out of nine instances. In those cases in which cardiac hypertrophy could be demonstrated this factor was within normal limits in five out of six instances. The percentage of the pulse-pressure formed by the second phase was below 40, or not obtainable in ten out of fifteen cases.

III.—High Pressure Cases.

The following cases at the first examination showed a systolic pressure higher than normal : 266, 270, 329, 346, 368, 387, 396, 432, 479, 506, 524, 543, 566, and 637 (Table III).

Table III.—High Systolic Pressure.

Case	Sex	Age	Systolic	Diastolic	Pulse	C. E.	Overload	Per cent., second phase	C. S. : C. W. ratio	Time interval
						Per cent.	Per cent.			
*266	F.	59	152	40	112	73·0	230·0	30·3	51·1 : 48·2	—
			138	64	74	53·0	65·0	32·4	78·3 : 21·6	24 hours
*270	F.	48	184	98	86	46·0	37·0	23·2	65·0 : 34·8	—
329	F.	23	134	66	68	50·0	53·0	25·0	65·8 : 34·0	—
346	F.	54	*244	130	114	46·0	37·0	52·6	87·6 : 12·2	—
			182	105	77	42·0	23·0	11·6	89·5 : 10·2	1 month
			182	102	80	43·0	28·0	27·5	65·0 : 35·0	3½ months
			196	105	91	46·0	36·0	16·4	—	2½ months
			182	105	77	42·0	23·0	—	—	6 months
			210	105	105	50·0	50·0	12·3	74·2 : 25·6	7 months
			215	95	120	55·0	76·0	20·8	79·9 : 19·9	1 month
			189	100	89	47·0	39·0	11·2	—	1 month
368	F.	19	142	56	56	53·0	50·0	31·5	81·5 : 31·5	—
387	F.	39	140	82	58	41·0	20·0	37·9	72·3 : 27·4	—
396	F.	40	*180	48	132	53·0	225·0	21·2	74·2 : 25 7	—
			*154	82	72	46·0	37·0	27·7	—	43 days
			*144	76	68	47·0	39·0	32·5	76·4 : 23·5	4 months
			164	90	74	45·0	32 0	6·7	—	6 months
			170	90	80	47·0	38·0	23·7	83·7 : 16·2	5 months
432	F.	32	174	98	76	43·0	27·0	42·1	84·2 : 15·7	—
			148	86	62	41·0	22·0	29·0	67·7 : 32·2	5 weeks
479	F.	43	215	120	95	44·0	29·0	—	—	—
			*206	130	76	36·0	8·0	36·8	86·8 : 13·0	1 week
			*204	118	86	42·0	22·0	6·9	—	9 days
			*198	120	78	39·0	15·0	15·3	—	3 days
			*196	112	84	42·0	25·0	—	—	4 days
			*196	114	82	41·0	21·0	31·7	87·7 : 12·1	1 week
			*176	124	52	29·0	−9·0	—	—	5 days

TABLE III.—HIGH SYSTOLIC PRESSURE (*continued*).

Case	Sex	Age	Systolic	Diastolic	Pulse	C. E.	Overload	Per cent., second phase	C. S. : C. W. ratio	Time interval
						Per cent.	Per cent.			
			*200	128	72	36·0	6·0	—	—	After walking
			185	102	83	44·0	31·0	3·6	—	11 days
			185	109	76	41·0	19·0	6·5	85·4 : 14·4	2 weeks
			176	105	71	40·0	17·0	—	—	3 weeks
			155	95	60	38·0	13·0	50·0	83·3 : 16 6	1 month
			185	105	80	43·0	26·0	31·2	81·2 : 18·7	3 months
			200	119	81	40·0	18·0	9·8	86·3 : 13·5	5 months
			194	112	82	42·0	23·0	23·1	—	15 days
			195	105	90	46·0	35·0	—	—	2 weeks
506	F.	54	170	60	110	64·0	133·0	12·7	59·9 : 39 9	—
			152	80	72	47·0	40·0	8·3	83·3 : 16·6	1 week
			154	90	64	41·0	21·0	—	—	5 weeks
			152	86	66	43·0	26·0	18·1	75·6 : 24·1	2 weeks
			150	80	70	46·0	37·0	—	—	3 weeks
524	M.	61	184	95	89	48·0	43·0	20·2	81·9 : 17·9	—
			142	75	67	47·0	39·0	37·3	67·1 : 32·8	10 weeks
			142	80	62	43·0	27·0	24·1	88·6 : 11·2	3 weeks
			145	76	69	47·0	40·0	17·3	79·6 : 20·1	1 month
			127	65	62	48·0	45·0	40·3	72·5 : 27·3	1 month
			135	65	70	51·0	57·0	20·0	77·1 : 22·8	17 days
			155	75	80	51·0	56·0	17·5	85·0 : 14·9	2 months
			162	85	77	47·0	40·0	16·8	88·2 : 11·5	13 days
			165	90	75	40·0	33·0	17·3	83·9 : 15·9	1 week
			155	75	80	51·0	56·0	43 7	78·7 : 21·2	3 weeks
			144	87	57	39·0	15·0	31·5	77·1 : 22·7	1 month
			*166	90	76	45·0	34·0	13·1	68·3 : 31·5	25 days
543	F.	47	*194	68	126	64·0	135·0	39·6	79·2 : 20·6	—
			*188	86	102	54·0	68·0	3·9	88·2 : 11·7	3 weeks
			*156	74	82	52·0	60·0	29·2	77·9 : 21·8	1 week
			*154	76	78	50·0	52·0	2·5	79·4 : 20·4	1 week
			137	62	75	54 0	70·0	—	—	1 week
			165	60	105	63·0	125·0	17·1	71·3 : 28·5	19 days
			*158	76	82	52·0	57·0	—	—	24 hours
			*160	80	80	50·0	50·0	27·5	82·5 : 17·5	8 days
			†*152	86	66	43·0	26·0	18·1	—	10 days
			*170	80	90	52·0	62·0	17·7	84·3 : 15·5	1 week
			*174	90	84	48·0	43·0	—	—	8 days
			*158	86	74	45·0	33·0	—	—	8 days
			155	65	90	58·0	88·0	—	—	2 weeks
			155	70	85	54·0	51·0	—	—	3 weeks
			134	58	76	56·0	81·0	—	—	4 weeks
			160	60	100	62·0	50·0	—	—	7 weeks
			174	60	114	65·0	140·0	—	—	2 months
			*182	70	112	61·0	110·0	17·8	80·3 : 19·5	2 weeks
			*162	60	102	63·0	120·0	—	—	6 days
			*192	80	112	58·0	90·0	—	—	2 weeks
566	F.	35	*146	100	46	31·0	−4·0	39·1	—	—
637	M.	57	148	75	73	49·0	27·0	17·8	82·1 : 17·7	—

† First point absent.

Case 266 presented an arrhythmia. All of the cases presented clinical evidence of cardiac hypertrophy except Cases 387, 524, and 566. In these cases at the first examination the pulse-pressure was above 40 in all. The cardiac efficiency factor was above 35 in thirteen; in one it was 31 per cent. The overload was above 100 per cent. in four; between 50 and 100 in two; below 50 in seven and a minus quantity in one. The second phase of the auscultatory blood-pressure determination was 40 per cent. or more in four cases, and could not be determined or was below 40 per cent. in ten cases. The C.S. : C.W. factor was on the side of cardiac strength in twelve cases, and was unobtainable in two cases. In two of the cases while the patient was under treatment the systolic blood-pressure gradually returned to normal limits, namely, Cases 266 and 543; but in the latter case it again became high. It appears to be the rule, however, that in a case in which a high systolic pressure is determined at the first examination such organic changes have taken place in the heart, kidneys, or arterial system, that it is impossible to restore a normal condition to the circulation.

Case 266 apparently shows a reduction of systolic pressure under treatment. When the patient was first seen she had an arrhythmia, which was diagnosed by clinical methods as auricular fibrillation, and a systolic pressure of 152 mm.; pulse-pressure, 112; cardiac efficiency, 73 per cent.; overload, 230 per cent. In twenty-four hours without treatment, except rest in bed and a cathartic, her systolic pressure was 138 mm.; pulse-pressure, 74; cardiac efficiency, 53 per cent.; and overload, 65 per cent. The patient then passed out of my immediate care, although the subsequent history was that she was operated upon first one month later, when the right superior thyroid artery was ligated and cut, and a second time, three months after the first operation, when the

left superior thyroid artery was ligated and cut. Both operations were done by Dr. M. B. Tinker. The patient died suddenly nine days after the second operation, probably on account of the auricular fibrillation.

Case 543 was a female, aged 47 years. When first seen she had a systolic blood-pressure of 194 mm., a pulse-pressure of 126, a cardiac efficiency of 64 per cent., and an overload of 135 per cent. She was under my care in the Rochester General Hospital for about six weeks, when she was allowed to go home. By that time her systolic pressure had fallen to 154 mm., pulse-pressure to 78, cardiac efficiency to 50 per cent., and cardiac overload to 52 per cent. A week later her systolic pressure was 137 mm., pulse-pressure 75, cardiac efficiency 54 per cent. About two weeks after that she began to present symptoms of a recrudescence, her systolic pressure had increased to 165 mm., pulse-pressure to 105, cardiac efficiency to 63 per cent., and overload to 125 per cent. She returned to the hospital, where she was under treatment twenty-five days. Her systolic pressure was still 175 mm., pulse-pressure 90, cardiac efficiency 52 per cent. During the next four months her condition improved steadily, and at the end of that time her systolic pressure was 134 mm., pulse-pressure 76, cardiac efficiency 56 per cent., and overload 81 per cent. The next month, however, the systolic pressure had returned to 160 mm. Later, coincidently with a coryza, she had an increase in her symptoms and the systolic pressure rose to 182 mm., pulse-pressure to 112, cardiac efficiency to 61 per cent. This patient has had albuminuria from time to time, but casts have never been found.

In the cases in which the systolic pressure was higher than normal, the pulse-pressure was above 40 in all. The cardiac efficiency factor was above normal in thirteen out of fourteen cases. The overload was above 50 per cent. in six out of fourteen cases. The percentage of the pulse-pressure formed by the second phase was below 40 in ten out of fourteen cases. It appears to be the rule that in a case in

which a high systolic pressure is determined at the first examination such organic changes have taken place in the heart, kidneys, or arterial system that it is impossible to restore a normal condition to the circulation.

In *Case* 529 I was able to study the blood-pressure during hemithyroidectomy, performed by Dr. D. G. Hastings in the Rochester General Hospital. The patient was a female, aged 38 years, who was employed as an auditor in one of the department stores. When she was first seen she had had thyroid gland disease for about three years, and complained of nervousness, headache, dyspnœa, palpitation of the heart, pounding in the chest, a sensation of crawling on the skin, and flashes of heat. The physical examination showed cutaneous erythema, exophthalmos, dermographia, cyanosis, tremor of the tongue, slight hypertrophy of the heart, accidental murmurs, palpable liver edge, rapid pulse (140), pyorrhœa alveolaris, and high palatine arch. She was admitted to the Rochester General Hospital in May, 1914. After four weeks of hygienic treatment with medication the systolic blood-pressure was 148 mm.; diastolic pressure, 90 mm.; pulse-pressure, 58; cardiac efficiency, 39 per cent.; second phase, 44·8 per cent.; C.S., C.W., 68·9 : 30·1; overload, 14 per cent. She was allowed to leave the hospital at the end of six weeks, improved in all of her symptoms, except the tachycardia. Three weeks later she had a nervous attack with diarrhœa, and her pulse-rate was again 140 per minute. She was readmitted to the Rochester General Hospital in August, at which time the following blood-pressure observations were recorded: Systolic, 139; diastolic, 60; pulse-pressure, 79; cardiac efficiency, 56 per cent.; second phase, 12·6 per cent.; C.S., C.W., 50·4 : 49·3; overload, 81 per cent.

The right lobe and one half the isthmus were removed by Dr. Hastings, under ether anæsthesia, on August 13. Before the operation systolic pressure, 158 mm.; diastolic, 98; pulse-pressure, 60; cardiac efficiency, 37 per cent.; second phase, 26·6 per cent.; C.S., C.W. ratio not obtainable; overload, 11 per cent. The cuff of a Tycos sphygmomanometer was applied to the left arm as soon as anæsthesia was complete, and blood-pressure observations were made every five minutes during the operation. The systolic

pressure had risen to 180 mm. after etherization, and remained high until, following a rather sharp venous hæmorrhage, it fell to 166 mm. It then continued to fall, and after the gland was removed and the closure of the wound was being attended to it fell to 124 mm. Then 1 c.c. camphorated oil was injected and one gallon of oxygen was given, followed by a gradual rise to 134 mm. The camphorated oil was then repeated, and 500 c.c. normal saline were given by hypodermoclysis. By 5.35 p.m. of the day of operation the systolic pressure had returned to 156 mm. The following day it had reached 170 mm., and it continued high for two days. Then, following the administration of sodium nitrite, gr. i, t.d., it gradually fell to 140 mm., when the sodium nitrite was stopped. When the patient left the hospital on September 7 the systolic pressure was 126. After two months spent in a convalescent home at Bath it was 131 mm.

The diastolic pressure fell from 100 to 90 when the operation was begun. It rose when the systolic pressure fell, so that when the first dose of camphorated oil was given it was 102 mm. After the oxygen inhalation it fell to 60 mm., to rise to 76 mm. after the hypodermoclysis. Following the administration of sodium nitrite it fell to 30 mm., and fluctuated markedly while this drug was given. At the last observation the diastolic pressure was 74 mm. The pulse-pressure rose after the etherization was completed, but gradually fell to 24 just after the gland was removed. After the administration of the camphorated oil and oxygen it rose gradually, and continued to rise until after the sodium nitrite was begun, when it began to fall, and finally registered 59 mm.

It would seem that the manipulation of the thyroid body in hemithyroidectomies is followed by a fall in systolic pressure, a rise in diastolic pressure, and a fall in pulse-pressure. In this case the administration of camphorated oil, oxygen, and hypodermoclysis was accompanied by a rise in the systolic pressure, a fall in the diastolic pressure, and a gradual rise of the pulse-pressure. In this case the administration of sodium nitrite by mouth was followed by a fall of the systolic pressure, a fall of the diastolic pressure, and a fall of the pulse-pressure.

I saw this patient nine months after her operation. She is apparently well. Her systolic blood-pressure observation made in the recumbent posture with a Riva Rocci instrument is as follows :—

First point	135	Pulse-pressure	67
Second point	125	Cardiac efficiency ...	49 per cent.
Third point	97 ; 41·7 per cent.	Cardiac overload ...	48 per cent.
Fourth point	70	C.S., C.W., ...	81·9 : 17·8
Fifth point...	68		
Pulse ...	118, recumbent		

The pulse-pressure is still high, the cardiac efficiency is above normal, and the overload is high. While the patient is fairly well and comfortable, she can by no means be thought, clinically, to have a normal circulation.

An interesting question arises which cannot, of course, be settled by the observation of a single case. Are the fluctuations of blood-pressure due to the operation *per se* or to the manipulation of the thyroid gland? It would seem that the fall of systolic pressure may be attributed to the forcing of thyroid secretion into the vessels by the manipulation of the thyroid body. This corresponds to the influence of the intravenous injection of thyroid extracts into dogs reported by Sanford and Blackford.

Table IV shows the influence of an acute infection and rest in bed on one of the high pressure cases.

The patient was a male, aged 61 years, whose systolic pressure at the first examination was 184 mm. During a course of treatment his systolic pressure registered at one time as low as 127 mm., but just before the acute infection began in March, 1915, the systolic pressure was 166 mm. The acute infection was ushered in by distension of the stomach, tired feeling, a temperature of 101° F., and numerous extrasystoles. After twenty-four hours in bed the systolic pressure was 146 mm. The patient had a typical

influenzal attack with laryngitis and accompanied by bilateral purulent otitis media, necessitating the puncture of both ear drums. During this acute infection and resulting confinement to bed the patient's systolic pressure was constantly below 150 mm., except on one occasion, when it was 158 mm., following the puncture of one of the ear drums. The pulse-pressure, the cardiac efficiency, and percentage of the pulse-pressure formed by the second phase showed no very great fluctuations from figures obtained before the acute infection began. The cardiac overload varied from 13 per cent. to 39 per cent.

TABLE IV.

Case	Sex	Age	Systolic	Diastolic	Pulse	C. E.	Overload	Per cent., second phase	C. S. : C. W. ratio	Time interval
						Per cent.	Per cent.			
*524	M.	61	166	90	76	45	34	13·1	68·3:31·5	—
			146	80	66	45	32	21·2	75·7:24·2	24 hours
			138	76	62	44	31	25·8	80·6:19·3	,,
			148	78	70	47	39	17·1	79·9:19·9	,,
			138	80	58	42	21	24·1	75·8:24·0	,,
			144	88	56	38	13	14·2	64·2:35·6	,,
			148	80	68	45	35	29·4	70·5:29·3	,,
			146	80	66	45	35	6·3	81·7:18·0	,,
			130	74	56	43	25	21·4	71·4:28·5	,,
			138	84	54	39	14	7·4	66·6:33·3	,,
			136	78	58	42	24	31·0	68·9:30·9	,,
			136	78	58	42	24	24·1	65·4:34·4	,,
			144	86	58	40	17	10·3	—	,,
			142	80	62	43	27	35·4	80·5:19·3	,,
			158	84	74	46	38	18·9	70·2:29·7	,,
			130	78	52	40	16	30·7	88·3:11·4	,,

CONCLUSIONS.

From the study of these cases it seems legitimate to conclude that the first effect of dysthyroidism on the blood-pressure is to produce a lowering of the systolic pressure. This is accompanied by a disturbance in the pulse-pressure, usually an increase. After the case has persisted for a varying period the systolic blood-pressure begins to rise, and,

after the development of cardiac hypertrophy and renal and vascular changes, the cases are converted into typical examples of chronic hypertension.

The study of the various functional myocardial tests would seem to show that the myocardium is disturbed in nearly all cases, whether or not there is clinical evidence of such disturbance.

During the operation of hemithyroidectomy in one case, there was a sharp fall of systolic and pulse-pressure and a rise in diastolic pressure. After the operation the systolic pressure again rose to a high point, but after the use of sodium nitrite it returned to a more nearly normal figure.

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DISCUSSION.

Dr. W. Jarvis Barlow (Los Angeles, Cal.): I am glad somebody else has the feeling I have. Especially with those who go first to a new physician the pressure will go down after a little rest and confidence. As to the matter of operation that Dr. Swan has spoken of, that we cannot judge from one case. But the suggestion he has given us I do not think bears out at all with the operation of the thyroid gland, because you see the same thing in any other operation.

With regard to the dysthyroidism and blood-pressure, my experience has borne out, I think, what he said, that when you have high blood-pressure it is due to secondary cardio-vascular changes. I have seen two cases recently having high blood-pressure, not due to the

dysthyroidism, but to the cardio-vascular changes secondary to the primary condition.

Dr. PHILIP KING BROWN: May I ask if Dr. Swan has had any experience in the over-activity or persistence of the thymus gland in relation to this matter?

Dr. JOHN A. LICHTY (Pittsburgh, Pa.): Dr. Swan's paper is interesting from many standpoints. Some points are entirely in accord with my own experience. I do not say that my own experience is sufficient to draw the conclusions which Dr. Swan suggested. For instance, the effect of operations upon blood-pressure, especially so in a case of fibroid of the uterus, where there was a very high blood-pressure, systolic and diastolic. The patient had a great deal of care and consideration on account of it. After the operation it was very low while lying in bed, and later when going about went back to the usual condition. Here we have the points as to the effect of rest in bed, effect of the operation, and the effect of a panhysterectomy to consider.

There is also a peculiar condition which I have noticed in taking blood-pressure, which might have something to do with the results which Dr. Swan presents, I refer to the blood-pressure taken at the first consultation. Many a patient who comes into the office for the first consultation will have a certain blood-pressure, which in a few days after becoming acquainted with the physician may be quite different. This also happens sometimes when patients come a long distance, perhaps as the effect of the journey. There are so many factors, it is really a dangerous thing, in my mind, to draw any conclusions. I think the line in which Dr. Swan has approached this subject is to be commended.

Dr. JOHN M. SWAN: In the first place, Mr. President, in answering your own question about the meaning of dysthyroidism, it is necessary for me to say that for the last three and a half years I have been living in a thyroid country. I noticed many young women on the street with pyramidal-shaped necks, indicating a definite thyroid enlargement. Suddenly it dawned upon me that the region in which I live is in the Great Lakes region. I have come to the conclusion that it is impossible to separate hyperthyroidism from hypothyroidism. I think hyperthyroidism has the same relation to exophthalmic goitre, for instance, that tuberculosis has to consumption. I think you will all admit that a person usually has tuberculosis a great many years before he has consumption, except perhaps in the acute miliary cases and the cases of acute pneumonic phthisis. I am going to change my mind if necessary, but at present I think that there are disturbances of the thyroid apparatus that begin early in life and are accompanied by nervous phenomena, and other phenomena which it would take too long to go into, which finally develop into true cases of exophthalmic goitre on the one hand, or into typical cases of hypothyroidism on the other hand. I think the best name for this condition, therefore, is dysthyroidism.

In regard to the question concerning the diastolic blood-pressure, I never have any trouble. I have been using the auscultatory method since 1910, and always read my diastolic pressure at the point at which all sounds disappear. In examining a patient the blood-pressure observation is the last thing I do. I always make it in the recumbent position. I always put the cuff on the bare arm, and always use the same stethoscope. I have the conditions as nearly the same as possible at every observation, so that the results recorded are open as little as possible to the criticism that Dr. Lichty has made.

I am glad Dr. Lichty made the statement about the effect of operations on blood-pressure. I have tried to find in the literature some published figures of observations made during operations, but so far have been able to find none. The question occurred to me, as I said in my paper, is the fluctuation of blood-pressure that occurred in the case reported, the result of the anæsthetic, the result of the operation *per se*, or the result of both plus handling the thyroid gland and the consequent squeezing of the thyroid secretion into the circulation? Of course, I cannot tell.

In regard to Dr. Barlow's statement concerning the cases of high pressure due to secondary cardio-vascular changes, I believe he is right. I think one result of continual dysthyroidism is to produce a high blood-pressure. I frequently see cases of chronic cardio-vascular disease without explanatory previous history. I find cases in which none of the acute infections have occurred, in people who have lived exemplary lives, who have not abused tobacco or alcohol, particularly women in the higher walks of life, in whom the influence of alcohol and intemperate eating, and tea and coffee addiction, and all that sort of thing, can be definitely eliminated. In such cases I sometimes find evidence of thyroid disturbance, and my idea is that this disturbance has been going on for years. It started, perhaps, when they were young girls, made them nervous, gave them a low blood-pressure and certain tremors. With the progress of time the continued action of the abnormal thyroid secretion on the cardio-vascular system has resulted in the same changes that follow the acute infections, alcoholism, the over-use of tobacco, the over-use of proteid food, &c. This is an idea which may not be correct, but which, it seems to me, is worth while calling forth serious consideration.

In regard to Dr. Brown's question: No, I have had no experience with thymus. Unfortunately, autopsies are not popular in my locality. Among the patients I see consent for autopsy is not readily obtainable. I have, of course, the relation in mind, and hope to be able some day to make some contribution to its solution. From the literature I am inclined to think that when dyspnœa is a prominent symptom thymus disturbance is probably present.

HELIOTHERAPY IN COLORADO.

By CHARLES FOX GARDINER, M.D.
COLORADO SPRINGS, COLORADO.

[READ BY TITLE.]

The action of sun baths in treating disease is now fairly well understood by many physicians, thanks to a number of excellent articles which have lately appeared in several medical journals. However, a brief description of the scientific facts now known in regard to the action of the solar energy upon the human body may be helpful.

The rays of the sun falling on the skin of one of the white race produce first a congestion, followed by an irritation or burn if continued long enough. This first congestion, if not carried to the point of irritation or destruction of the outer layer of skin, is of benefit to the body in several ways. The cutaneous vessels are dilated, internal congestion is relieved, the general circulation of blood and lymph is stimulated, the heart's action assisted and the kidneys and skin are encouraged to act more rapidly. In this way the metabolism of the body becomes more active, as both the absorptive and eliminative powers of the cell are increased.

As the heat rays falling on the skin produce an increased flow of blood to the part exposed, antibodies that combat disease are formed in excess of the normal, hæmoglobin is increased and the blood count shows an increase in the

FIG. 1.—Heliotherapy. Advanced case with disease of both lungs and spine.

FIG. 2.—Albino taking first sun bath.

FIG. 3.—Cases treated by heliotherapy in Colorado. From left to right, knee-joint, kidney, spine.

FIG. 4.—Heliotherapy in Colorado. Tuberculosis of the knee-joint.

number of red cells. These effects can often be observed by the use of different baths or massage, which tend also to cause a superficial congestion; but in using the solar energy on the skin there is another force which, perhaps, is more potent and far-reaching in its effect on man than all the other rays together, and these are the actinic, or ultra-violet rays, called also the dark or chemical rays. Although these rays from the sun cannot be seen, they exist in the sunshine and have a powerful effect on all cellular life.

The actinic rays falling on a white skin are nearly all reflected back and do not penetrate, but when the white skin becomes bronzed from deposit of pigment this pigment acts as a transformer to the actinic rays and they then pass freely into the skin. The actinic rays now allowed to penetrate into or through the skin act on the corpuscles of the blood, present in large numbers in the dilated cutaneous vessels, stimulating and vitalizing them before they are carried by the general circulation to all organs and tissues of the body.

Another condition present during the treatment by sun baths, and one often overlooked, is the reflex action induced by air moving on the surface of the body. This reflex, or stimulation of the nerves of the skin, is a factor of much importance, and affects metabolism in a decided manner, as our skin, having been protected by clothing for many generations, has become physiologically inert, and the air bath is of itself of much assistance in giving heliotherapy, and like it a tonic to the body, possibly because in the "out-door cure," in giving fresh air to the lungs and sunlight and air to the skin, we are merely returning to the primitive environment of the human race.

In using heliotherapy in Colorado there are some climatic conditions that have a more or less important bearing on the therapeutics of sun baths. At an elevation of a mile above

the sea-level, Colorado has a dry, thin air. The sunshine being a very constant factor, the treatment can be given in a regular and systematic manner, without the annoying interruptions of cloud, fog, or rain, so common in many damp localities that they often interfere for days at a time with the daily use of sunshine.

The rays that constitute the effective force in the sunshine, the actinic rays, are all absorbed by a dense atmosphere, so that over cities and in the damp lowlands in general these valuable rays do not reach the patient but are nearly all dissipated by passing through thick and humid air, while in dry, thin air, such as Colorado possesses, the actinic rays pass quite freely to the patient and exert their curative effect. Thus, the "dose" of sunlight in Colorado can be given regularly, the dose is more efficient in a given time on account of the presence of actinic rays, and, moreover, the dry air surrounding a patient while taking a sun bath prevents the enervating effects due to humid heat, at the same time allowing a free exposure during cold weather without chill or danger.

In treating tuberculosis by heliotherapy the brilliant results obtained in bone and joint tuberculosis have rather cast into the shade the results of this method in pulmonary tuberculosis. The comparison, however, is not a fair one, as in one form of the disease the invasion is local and can often be arrested by drainage, with proper apparatus to insure rest; while, in the other, vital organs are attacked, poisons are easily taken up by the blood, rest is difficult to accomplish, drainage is often imperfect and metabolism disturbed, so that we cannot reasonably expect as good results from heliotherapy in pulmonary tuberculosis as in tubercular bones or joints, and the comparison between the two conditions is not a fair one.

If the patients with pulmonary tuberculosis are given the benefit of careful selection, and heliotherapy is used with patience and caution, not expecting rapid results, it will no doubt be found that the sun bath is a measure well worth the trial. The directions to patients are as follows:—

Protect head and neck by shade of building or a white hat.

Wear amber-coloured glasses.

Use reclining chair or couch.

Have a free circulation of air but avoid cold winds.

The hours between 11 a.m. and 3 p.m. are the best.

First day expose feet and legs to knees for five minutes.

Second day the same.

Third day for ten minutes if skin is not hot or inflamed.

Have physician see skin before taking more baths.

Gradually increase the area exposed to the sun.

First legs, then abdomen, then chest and arms, turning over so as to expose back also.

Take temperature before and after bath. If there is elevation of temperature, headache, nervousness, or fatigue, report to your physician before taking another bath.

As to the selection of patients with pulmonary tuberculosis for sun treatment, there is apparently no rule that can guide us in all cases. There are undoubtedly certain individuals who will not react favourably to such treatment, who will, in spite of the utmost care and caution, have irritation of the nervous system, headache, restlessness, or rapid pulse and temperature after a brief exposure. This can be judged only by a trial.

There are two types among the tubercular invalids that are supposed to be most unfavourable subjects for sun treatments: those with advanced disease of some years' standing who are having daily temperature above normal, and the blondes who have thin, white skins and form blisters instead

of pigment in the skin from sun exposure. In the first class I have a patient who had been in Colorado for three years, lived in the open air day and night, was carefully watched, given proper food, and was completely at rest. There was involvement of both lungs with cavity formation, sputa 6 oz. in twenty-four hours, tubercle bacilli present, rapid pulse, afternoon temperature 101° F., loss of weight 20 lb., tubercular spine with deformity, a large abscess holding about 2 quarts on left hip, due to the spinal disease, much pain. The condition had gradually grown worse from year to year. No change was made in food, air or rest, but heliotherapy was tried. As the illustration shows, the body, after eight months of exposure, is dark bronze in colour. The physical signs show fibrosis and retraction in both lungs; sputa 1 oz. in twenty-four hours; pulse 80; temperature normal; gain in weight 16 lb.; abscess from spine absorbed without opening; no pain on walking; spine fixed at point of deformity. It is hard to believe that the sun baths did not produce the change for the better in this patient's condition, because it was the only change made in her treatment after three years of all that climate and sanatorium treatment could do for her disease.

In regard to the thin-skinned, blonde type, I have under treatment a gentleman of 34 years with an extensive involvement of left lung, small cavity at apex, who has been ill for one and a half years; loss of flesh 16 lb., sputa 4 oz. in twenty-four hours, streaked with blood, tubercle bacilli present, afternoon temperature 101° F. The X-ray confirmed the physical examination. This patient is really an albino with perfectly white hair, very white and thin skin that burns easily from slight exposure to the sun, eyes intolerant to light. There being but little improvement in his condition after some weeks in Colorado, I decided to try

heliotherapy, although such an extreme instance of the blonde type seemed unfavourable to the experiment. I therefore used more caution than usual, protected the head by deep shade, the eyes by amber glasses, had the pulse and temperature taken both before and after the bath. I exposed the feet only for five minutes the first day, then the lower part of the legs for five minutes the next day, when the skin became hot and tender I waited a day or two for this to subside, never allowing the skin to become unduly inflamed, congested, or blistered, so that no desquamation occurred and no generally unfavourable reaction was noted. Gradually during three months the whole body, except the head, has been exposed. There has been no bronzing or pigment formed in the skin, but in its place a curious chronic congestion has occurred, dark red with a faint blue colour in places. This is not painful or hot, but it is constant and remains the same from day to day; but it gradually disappears after ten days when sun exposure is discontinued. This seems an effort of Nature to mass the blood on the surface in place of pigment, so that the skin is not white but dark red, and probably the actinic rays pass through this darkened skin as they do when pigment is present, thus, in the blonde types, reaching the blood corpuscles by this method without the pigment being present.

The change in this patient under heliotherapy has been most gratifying. The physical signs have improved; no moisture is heard over the lung, cough and expectoration have practically ceased; weight has increased 20 lb., and the temperature is normal. This improvement could have resulted from the usual climate and sanatorium methods employed without the use of heliotherapy, but the fact remains that a rapid and satisfactory improvement did take place in a blonde of extreme type while taking sun baths

every day, that the sun treatment was not injurious, but on the contrary, seemed to be of benefit, was not followed by unusual rise in temperature or pulse and no nervous symptoms developed, due to the exposure.

Conclusions.

Heliotherapy is now on a firm, scientific basis and will prove of value in treating pulmonary tuberculosis if used with caution and patience.

Advanced cases and those of the extreme blond type will occasionally respond favourably to sun baths.

In Colorado the sunshine can be used with regularity and the effect is enhanced by the large percentage of actinic rays, while the dry air on the skin tends to protect the body from heat exposure.

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THE CHOICE OF A CLIMATE IN CASES OF BRIGHT'S DISEASE AND NEPHRITIS.

By GUY HINSDALE, A.M., M.D.

HOT SPRINGS, VIRGINIA.

The questions are frequently asked: "What climate is best for a person with Bright's disease or nephritis?" and "In what way, for better or worse, does climate affect the kidneys?"

In a general way the impression seems to be that damp and cold climates are unfavourable and that warm and dry climates are beneficial; and that is probably true. For the last ten years I have been more or less interested in collecting data bearing on this subject so that it could be definitely shown just what localities might be advised and why they are beneficial.

One way of approaching this subject is by a study of mortality records, comparing those of different localities. The first criticism of this method is that "the registration of deaths gives a very imperfect view of the prevalence of disease. . . . It is fallacious to assume any fixed ratio between sickness and mortality." This is particularly true of infectious diseases, but the relationship is a little closer for the diseases we are considering. In the absence of morbidity records we have to rely on mortality statistics, and, of course, choose those coming from communities where there is some adequate system of registration.

And right here we meet with various difficulties. One of the first concerns the estimate of population. It is well known that in some communities there is an overwhelming desire to over-estimate population; this, of course, apparently lowers the death-rate, and in the absence of an accurate census it leads to irregularities in the ratios, independent of other factors in the case. In rapidly growing communities where the boom spirit prevails this should not be lost sight of. Then, again, the nature of the population makes a great difference. The more settled communities have a larger proportion of people over 50 years of age; but where migration is a factor in adding to the population, it averages younger, and chronic degenerative diseases are not so prevalent.

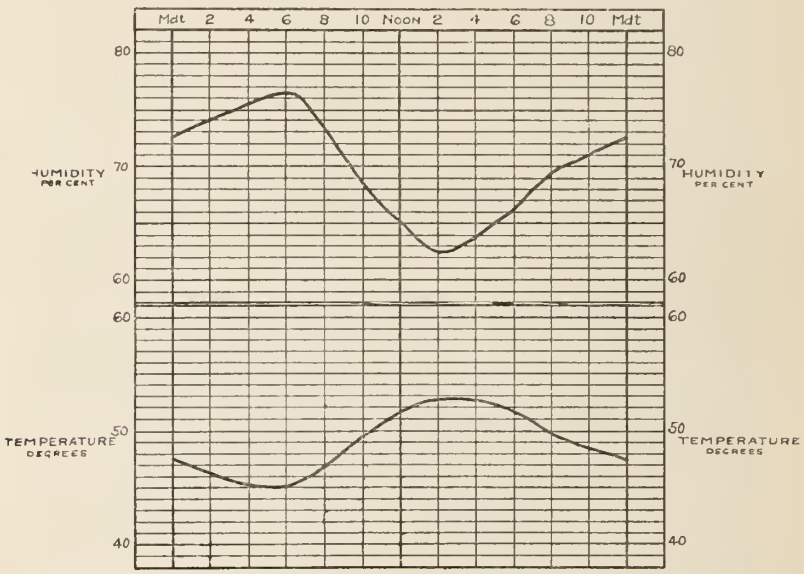
The occurrence of kidney disease doubtless has to do with personal habits, such as the use of alcohol, and with occupations such as mining, &c., with worry, hurried eating, especially of meats, and lack of exercise.

Without much intention to make a choice, excepting that the localities should be well distributed, I noted on three different years the combined mortality from Bright's disease and nephritis from statistics of twenty-five cities and obtained the following results, for which I am indebted to the various registrars of vital statistics.

I also obtained from the Chief of the United States Weather Bureau the relative humidity of the different stations as far as they were recorded. The average relative humidity is observed in the Government service at all stations here given at what is known as 75th meridian time, or at 8 a.m. and 8 p.m. Washington time. This is necessary for the service, but in practice it calls for some observations at 8 a.m. and for others at one, two, or three hours earlier in the day, according as the station is situated, where eastern,

central, mountain, or Pacific time is used. Two or three hours earlier in the morning tends to much higher records, and two or three hours earlier in the afternoon to lower humidity records, especially when the days are short.

The relative humidity, measured hour by hour, shows a wider diurnal variation than in the case of the temperature,

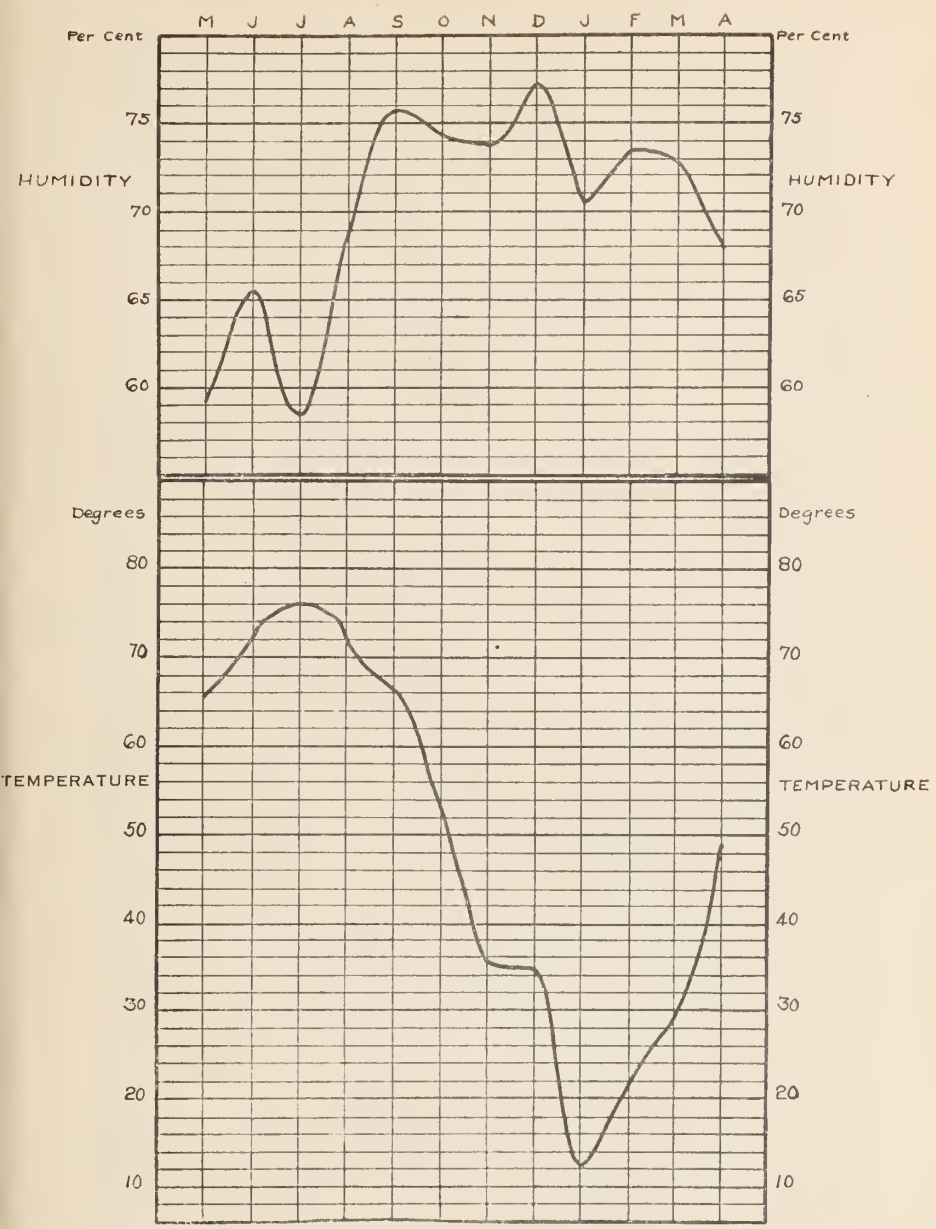


Relative humidity (bi-hourly), Chicago, from May, 1911, to April, 1912, inclusive, showing inverse relation to temperature.

as seen in the accompanying diagram for Chicago, which shows the inverse relation of the two curves.[1]

It will be noted that the cities here listed have been arranged for our purpose in the order of mortality for 1914, beginning with the highest and closing with that of lowest mortality. Some interesting features are apparent. In the first place, there is no obvious relation between mortality

[1] "Weather and Climate of Chicago," Cox and Armington. By courtesy of *Chicago University Press*.



Average relative humidity and temperature for Chicago, by months. Cox and Armington, "Weather and Climate of Chicago."

DEATHS FROM NEPHRITIS AND ACUTE BRIGHT'S DISEASE. RATE PER 100,000 LIVING.

	1904	1908	1914	PERCENTAGE OF RELATIVE HUMIDITY		
				8 a.m.	8 p.m.	Annual
Petersburg, Va.	243·0	302·6	320·0	—	—	—
Galveston, Texas	—	268·7	226·0	83	77	81
Ogdensburg, N.Y.	278·2	181·3	193·0	—	—	—
Albany, N.Y.	208·2	175·0	185·0	76	66	—
New Orleans	196·6	222·2	184·0	84	72	78
Baltimore	163·4	159·7	160·1	70	62	—
Philadelphia	160·0	160·7	153·5	75	65	—
Jacksonville, Fla.	200·0	195·3	142·5	81	73	80
Providence, R.I.	154·9	119·6	125·6	72	71	—
St. Joseph, Missouri	32·7	52·8	120·0	81	61	—
Newark, N.J.	140·0	122·7	114·2	—	—	—
Boston	79·6	89·4	110·0	72	70	—
San Francisco	123·2	75·0	109·0	86	69	80
Colorado Springs	80·0	75·9	106·0	—	—	—
Denver	114·3	136·7	102·2	67	43	51
New York City	174·5	145·7	101·0	70	61	73
San Antonio, Texas	82·0	124·5	90 0	86	58	—
Toledo, Ohio	67·0	69·1	89·0	78	68	—
Columbus, Ohio	54·0	78·3	84·8	80	65	—
Chicago	100·0	98·7	83·4	72	65	75
Portland, Oregon	58·0	73·8	69·0	—	—	87
St. Paul, Minn.	56·0	73·6	68·0	80	—	—
Pittsburgh, Pa.	55·0	66·7	65·6	74	61	—
Milwaukee, Wis.	58·5	64·4	63·7	76	70	—
Omaha, Neb.	64·1	56·3	55·3	78	60	70

statistics of these cities and the records of relative humidity as they are furnished. Another curious circumstance is that the mortality figures for Colorado Springs and Denver are nearest to those of New York, San Francisco, and Boston. Here we have two famous localities in the high and dry air of the Rocky Mountain region no better off, apparently, as far as mortality from acute Bright's disease and nephritis is concerned, than Boston, New York, and San Francisco at sea-level and subject to the characteristic climates of the Atlantic and Pacific seaboards. It seems to be true that

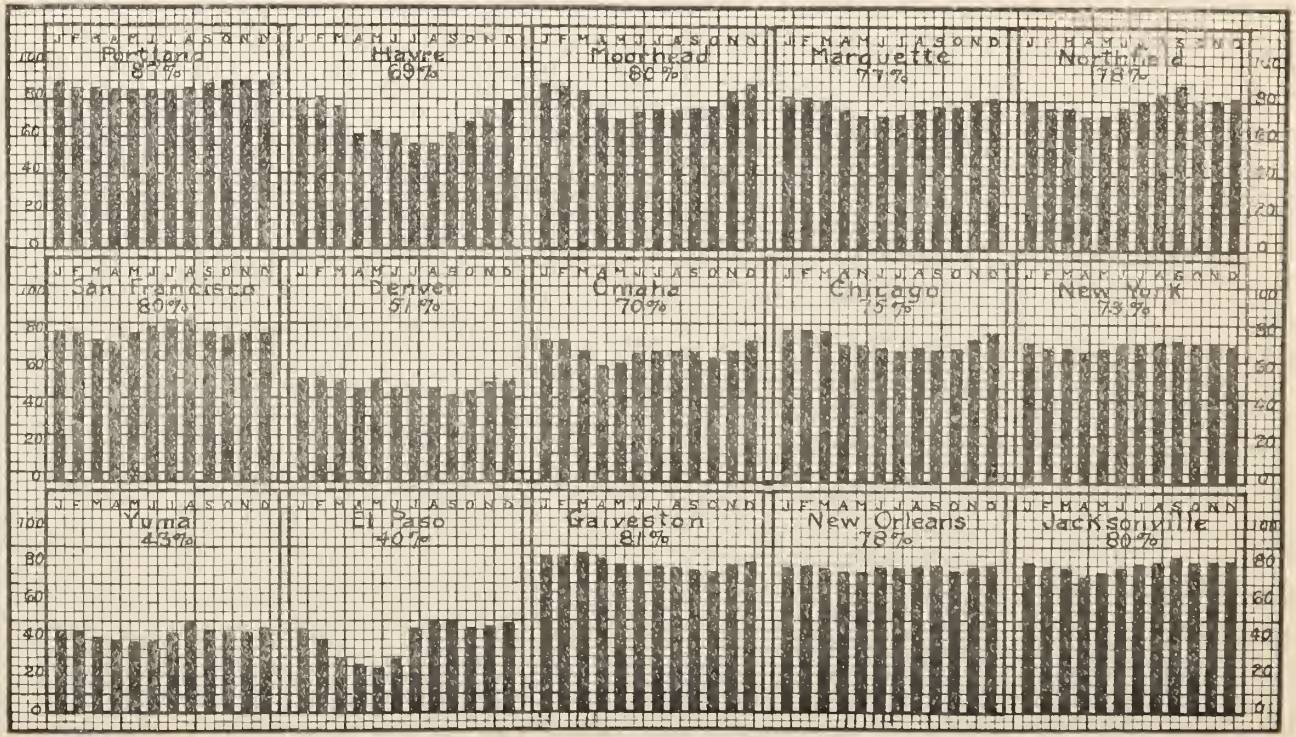
Omaha, Nebraska, among the cities mentioned, has the lowest death-rate from the diseases we are considering.

Another note may be made, and that is that "the old order changeth," and that as years pass few cities preserve a consistent position in the line.

Death-rate from Nephritis and Bright's Disease in States per 100,000 Population.

	1912	1913
Maryland	139·4	140·1
New York	138·5	134·7
Rhode Island	123·6	123·9
New Jersey	117·1	121·8
New Hampshire	121·7	118·8
Vermont	115·7	115·6
Connecticut	106·3	112·0
North Carolina	106·0	105·1
Pennsylvania	94·4	103·0
Maine	101·4	102·7
California	95·3	98·7
Missouri	98·4	95·2
Virginia	—	93·9
Indiana	92·9	92·7
Massachusetts	90·3	90·9
Ohio	90·1	87·6
Kentucky	75·4	80·3
Colorado	83·0	75·5
Michigan	75·4	74·9
Minnesota	70·6	72·5
Utah	69·6	64·7
Wisconsin	67·2	63·4
Montana	60·6	58·9
Washington	49·9	55·6

Through the courtesy of the University of Chicago Press I am able to show a chart illustrating the relative humidity by months in fifteen cities in the United States, arranged by Professors Cox and Armington. Now if we take the States that have adopted a system of registration, we find something more significant. In the accompanying table, taken from the United States Government mortality records, which I have arranged in the order of highest mortality for the year 1913, there is a decided grouping. The New



Average annual and monthly relative humidity in percentage for fifteen selected cities, 1889-1910. Cox and Armington.

England and Middle States, together with Maryland and North Carolina, fall in the upper division of high mortality, while the lower division of least mortality is almost exclusively made up from Central and Western States.

A glance at the accompanying table shows at once that the geographical grouping is marked. Perhaps we may see in this table some solution to our problem.

Turning now to clinical evidence as to climatic influence on acute and chronic kidney diseases we find some interesting observations which may lead to some practical conclusion. The most useful studies in this particular seem to have been made in Colorado and Texas.

Dr. Sewall has reviewed this whole subject in a careful paper which gives us some insight into the problems involved. Making his observations at Denver, elevation 5,280 ft., he has clinical evidence that cases of acute nephritis subside apparently as readily there as, judged by the literature, they might be expected to do at sea-level; and that cases of chronic nephritis may apparently recover or continue to live in comfort for years.

Dr. Sewall calls attention to the fact that the solid tissues, fluids and gases throughout the body are under a certain tension, which exactly balances the pressure of the atmosphere upon the surface. "It would seem to follow that when one enters a pneumatic chamber in which the air-pressure is lowered, or ascends from a lower to a higher altitude, there would be a movement of the labile (more movable) parts of the system from the interior towards the exterior. As has been said by others, it is as if the whole surface of the body, including the lungs and skin, were under a great cupping glass. There would be congestion of the skin through filling of the superficial vessels and, presumably, determination of blood to the air-passages and alveoli."

This cupping glass theory of the physiologic effect of diminished barometric pressure has a firm hold on the medical profession, but that it is erroneous for all ordinary conditions has been demonstrated. There is no doubt that with gradual ascent from a lower to a higher elevation external and internal pressures keep equal pace.[1] The view of Haldane and his colleagues, founded on experiences on Pike's Peak, Colorado, that all the physiological phenomena peculiar to high altitudes are directly or indirectly the result of lowered oxygen tension in the air must be depended upon to explain the facts.[2]

We all know that certain classes of persons, those who are rheumatic or gouty, are unpleasantly affected by a change in the weather. With the onset of bad weather, in other words, by a more or less marked fall in the barometric pressure, the onset of a cyclonic storm, the affected joints and muscles ache. Such persons are probably known to all of us and naturally excite our sympathy. Men who worked in caissons under abnormally high atmospheric pressure, measuring as much as thirty-eight or forty pounds to the square inch, in the railroad tunnels under the Hudson River and East River, suffered these pains in a marked degree. Hence unusual precautions were taken in restoring them to the usual atmospheric pressure. By means of a series of air "locks" during the period of decompression an equilibrium is gradually restored.

A fall of barometric pressure, as Dr. Sewall shows, can produce painful sensations in three ways: (1) The mere mechanical redistribution of the body fluids may excite, like a movement of massage, the nerve filaments—made abnormally sensitive by a chronic inflammation; (2) the accumu-

[1] *Cf.* Leonard Hill in "Recent and Further Advances in Biological Science."
[2] *Philosophical Transactions of the Royal Society.*

lation on the surface of blood containing an excess of intermediary (poisonous) metabolites may, as in gout, cause precipitation of irritative matters in the muscles or connective tissues; (3) the circulatory changes in the metabolic organs, particularly the liver, may so interfere with their oxidative powers as to lead to the abnormal accumulation of the products of intermediary metabolism.

As has been mentioned previously, when atmospheric pressure is lowered the venous system becomes engorged and the venous blood-pressure becomes abnormally high; and this seems to be true of the class of cases we have been considering.

Temperature Changes.—We know that a fall of temperature makes the peripheral blood-vessels contract, with a corresponding dilation of the deeper vessels. Relaxation of the superficial vessels is by no means so prompt as their contraction, and in cold weather active measures, such as friction and massage, may be required to overcome the stasis of the blood and lymph. Thus we see that the effect on the kidneys of a fall of temperature is to increase the volume of blood in the kidneys and to promote a rise of arterial tension by increasing the velocity of the blood flowing through them (and in all the deep-seated viscera) to increase their excretory activity and output.[1]

The associated cutaneous anæmia decreases the sweat and, as a consequence, the watery excretion of the skin devolves for the time being upon the kidneys, lungs, and intestines, and chiefly, as far as evidence goes, on the kidneys. Generally speaking, temperature rise produces the opposite effect of temperature fall, though not so quickly.

[1] For this and subsequent notes on the action of wind, temperature and humidity I am indebted to the excellent paper by Professor W. F. R. Phillips, of Charleston, S.C., in the TRANSACTIONS OF THE AMERICAN CLIMATOLOGICAL ASSOCIATION, 1906.

We know that when the bodily surface is exposed to cold the oxidizing capacity in the tissues thus cooled is lowered. When the extensive skin envelope is deprived to some extent of its oxidizing capacity by cold the body falls back on its heat-regulating apparatus to maintain its proper metabolism and a constant normal temperature of the blood. Increased oxidative capacity is therefore necessary elsewhere than at the periphery. It has been determined that a bath in water at 61° F. increases the oxygen absorption 47 per cent., while douches at this temperature cause an increase of oxygen absorption of about 110 per cent. This remarkable counter-effect is brought about by the heat-regulating mechanism. Metabolism is greatly stimulated by cold applications, especially when accompanied by active or passive exercise.

By analogy we may reasonably conclude that the same stimulus to the metabolic processes takes place in the presence of cold air as it does under the stimulus of cold-water applications, and, provided an adequate reaction occurs and the general circulation is maintained and the kidneys are unimpaired, the normal tissue changes go on in an uninterrupted and normal manner. When the available oxygen supply is large the increased metabolism is automatically and physiologically maintained.

It has been observed that the great majority of cases of uræmia occur in cold weather, and this would bear out the conclusion that faulty oxidation, which is primarily lowered by cold, is an important agent in causing uræmia, the kidneys in that case, as well as the heat-regulating apparatus, not being in a normal state of efficiency.[1] The primary cause of uræmia is a deficient oxidation of protein derivatives and the danger is in proportion of incompletely oxidized protein derivatives in the urine.

[1] See Ditman and Walker, *New York Medical Journal*, May 15, 22, 29, June 5, 1909.

Atmospheric Humidity.—The influence of atmospheric humidity upon the functions of the kidney is through its effect in promoting or retarding cutaneous and respiratory evaporation. With respect to the skin, evaporation from a moist surface depends, in a quiescent air, on the relative humidity. The nearer the point of saturation the slower the rate of evaporation, and the greater the deficit of saturation (low humidity) the more rapid the rate. So that in any given case the actual amount of water that may be evaporated will depend upon the temperature and on the wind movement. We see at once the great necessity of proper clothing that evaporation may not go on too rapidly. Professor Phillips states that in a quiet atmosphere at 30° F. and 80 per cent. relative humidity, a cubic foot of air could receive but ·038 of a grain of water more before becoming saturated; whereas at a temperature of 80° F. and the same relative humidity in the same time a cubic foot of air could take up ·219 of a grain, or about six times as much.

Wind.—There is yet one important element of climate that must be considered in this connection and that is wind. In connection with temperature and humidity, wind assumes a great importance when we consider the extent of our skin covering.

Now the wind, as we know, intensifies the action of temperature change. When, as is usually the case, the atmospheric temperature is lower than that of the body, the greater the velocity of the wind the greater the loss of heat, other conditions being equal. Cold winds can lower the temperature of the skin or of the body itself, as any of us can easily verify. If it is so in health, how much greater will be its effect when the body and its internal organs are diseased. And when we consider that in such subjects imperfect metabolic products are liable to accumulate in the

tissues as a result, or as a concomitant, of the blood lymph stasis already mentioned, we can understand that these effete products when thrown into the blood, upon restoration of normal circulation, may act as irritants to the kidney cells.

Soil.—Undoubtedly the character of the soil has much to do with the prevalence of nephritis just as in the prevalence of phthisis. A cold, damp, clayey soil, with sudden variations of temperature, is likely to chill the feet, and this co-operates with the bad effects of intemperance, overwork, and errors of diet which set on foot nephritis.

It is important to bear in mind that there is a distinctive difference between the false albuminuria of a cystitis or pyelitis and the true albuminuria of a nephritis. In Arizona, especially during the hotter season, it is not uncommon for persons to urinate only once or twice a day. The urine becomes concentrated and cystitis of a mild grade ensues and is followed by an increased desire to pass urine, though the quantity is small. The body-weight is reduced in the arid regions. Dr. Thomas Darlington, who formerly practised medicine in Arizona, ascertained that among fifty-eight persons of whom inquiry was made, only one had gained weight. Hence we see the improbability of persons with wasting diseases gaining weight in such a desiccating climate.

The southern portion of Arizona, which has a much more moderate altitude than the northern portion, southern California, or almost any part of the great interior valley of California, are desirable localities for persons with Bright's disease. So, also, are the interior stations in the Pacific slope and the Great Plains, including Nebraska, Montana, Colorado, and Utah.

One of the most brilliant examples of recovery from chronic kidney disease known to the writer followed removal

from an eastern city to San Diego, California. After a few years perfect health resulted and a most useful life was spared. It is now thirty years since, as an invalid, he left his eastern home, full of anxiety as to the future, which had in store for him only the richest reward.

San Diego has been said to have "the driest marine climate." It is a cool moist climate with much sunshine. The mean annual relative humidity is 75 per cent., varying from 67 per cent. in December to 80 per cent. in July and August.

Altitude.—Nephritis is not influenced unfavourably by high altitude. A great many cases of nephritis do remarkably well in Western Texas, in New Mexico, and along the slope of the Rocky Mountains, at five or six thousand feet elevation. Others do very badly.

In a recent communication from Dr. Edward C. Hill, of Denver, who has made many thousand urinary analyses during a long residence in that city, the opinion is expressed that true renal albuminuria is comparatively rare in Colorado, passive congestion from heart weakness and dilation being far more common. Personally, he has observed but a small number of cases of nephritis which could be directly attributed to alcohol. He states that acute nephritis, though not commonly observed, is exceptionally severe; chronic parenchymatous nephritis appears to terminate fatally in about the same time as at lower altitudes. The chronic interstitial type of the disease, on the other hand, is influenced favourably by this climate, and he attributes this to its tonic, invigorating qualities. Slight transient, circulatory albuminuria, due chiefly to high blood-pressure, is frequent in Denver. Of heterotoxic forms of albuminuria, that due to metallic poisons is seen rather frequently among the miners and about the smelters.[1]

[1] *Journ. Amer. Med. Assoc.*, 1905, p. 1173.

About ten or twelve years ago Dr. Wm. S. Carter,[1] Professor of Physiology in Galveston, Texas, made comparative observations at Boulder, Colorado, and Galveston, Texas, in an effort to ascertain what relationship, if any, exists between the temperature and humidity of these localities and the occurrence of Bright's disease.

Carter approached this subject by a series of experiments and observations on the consumption and elimination of water in these two localities. Galveston is at sea-level with a warm, and moist climate; Boulder has an elevation of 5,400 ft. (1,636 metres), and its climate is much dryer and cooler. The observations at Boulder were in July, 1899; those at Galveston were in the following September, and in the same month in 1903. The experiments were first made upon himself. By a strange coincidence the average output of urine in the six days in Boulder and in the nine days in Galveston, 1899, was exactly the same, viz., 1,404 c.c. daily. The average amount of water taken at Galveston was 2,140 c.c., and at Boulder 1,940 c.c. The temperature was rather cool for Galveston, but somewhat warmer than in the days at Boulder. Observations were also carried out at San Antonio, Texas, as well as at Galveston, on four individuals. The results showed that the average daily consumption of fluid was approximately 400 c.c. greater at Galveston than at San Antonio, 150 miles from the coast, and 700 ft. above sea-level, with a somewhat greater diurnal range of temperature and considerably lower relative humidity.

It is impossible to relate all the details of this excellent study, but it was evident in this, as in other similar climatic studies, that one takes more fluid as drink in a moist climate and passes less urine than in a dry climate. Dr. Carter remarks that the amount of moisture given off by the skin

[1] *Medical News*, November 12, 1904.

and the lungs in dry climates is not so great as is often thought. The sensible perspiration which results from the effort at heat regulation on the part of the animal economy in a hot, moist climate probably accounts for this difference. Dr. Carter was unable, in view of the statistics which were then available, to show any relation between Bright's disease and nephritis and relative humidity of the air.

Conclusion.

Bright's disease and nephritis are diseases clearly amenable to climatic treatment. Apparently the first requisite is that the air and soil shall be warm, sunny, reasonably dry, and free from malaria and disagreeable atmospheric changes. It is a disease attended by extensive degenerative changes in the renal and circulatory organs, and hence it is highly necessary to insure an abundant and undisturbed action of the skin. The locality should be conducive to an out-of-door life the year round. This is the well-known advantage of the climate of southern California, where the little rain that falls never does anyone any harm.

Our best course is to advise a warm climate first—one having moderate elevation and only a moderate rainfall, with small diurnal variations in temperature, the humidity being, perhaps, secondary in importance to temperature and wind.

The fact that other localities in the extreme north-west, such as the States of Washington and Montana, as well as Colorado, Minnesota, Wisconsin, Michigan, and Utah, have such diverse climates, and, nevertheless, fall on the favoured group, should not determine their choice as health resorts for renal cases, but would indicate some advantage in the character of the population and that the sum total of the elements of climate in those localities is conducive to a hardy race.

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